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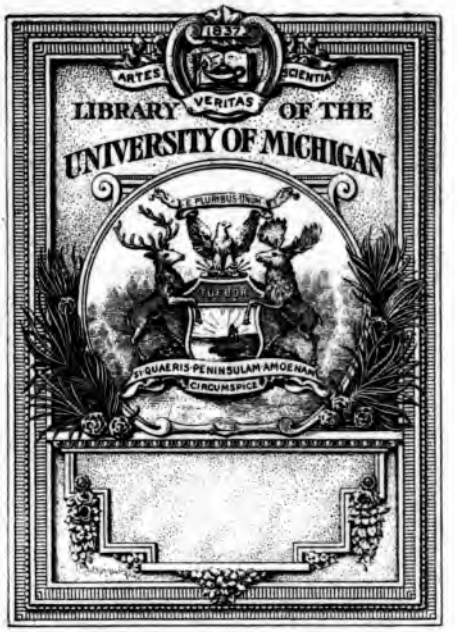
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FELLOWS OF THE SOCIETY APPOINTED BY THE COUNCIL AS REFEREES OF PAPERS,

FOR THE SESSION OF 1868-69.

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BARNES, ROBERT, M.D.
BARWELL, RICHARD.
BASHAM, WILLIAM RICHARD, M.D.
BURROWS, GEORGE, M.D., F.R.S.
CALLENDER, GEORGE WILLIAM.
CLARK, FREDERICK LE GROS.
COCK, EDWARD.
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MURCHISON, CHARLES, M.D., F.R.S.
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1805. WILLIAM SAUNDERS, M.D.
1808. MATTHEW BAILLIE, M.D.
1810. SIR HENRY HALFORD, Bart., M.D., G.C.H.
1813. SIR GILBERT BLANE, Bart., M.D.
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1817. WILLIAM BABINGTON, M.D.
1819. SIR ASTLEY PASTON COOPER, Bart., K.C.H., D.C.L.
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1825. GEORGE BIRKBECK, M.D.
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1861. BENJAMIN GUY BABINGTON, M.D.
1863. RICHARD PARTRIDGE.
1865. JAMES ALDERSON, M.D.
1867. SAMUEL SOLLY.

FELLOWS

OF THE

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L.—Librarian.	C.—Member of Council.

The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Sci. Com.* is attached to the names of those who have served on the Scientific Committees of the Society.

OCTOBER, 1868.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents, those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1841 *ABERCROMBIE, JAMES, M.D., Cape of Good Hope.

1846 *ABERCROMBIE, JOHN, M.D., Physician to the Cheltenham General Hospital, 13, Suffolk square, Cheltenham.

1851 *ACLAND, HENRY WENTWORTH, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Physician to the Radcliffe Infirmary; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847 ACOSTA, ELISHA, M.D., 24, Rue du Luxembourg, St. Honoré, Paris.

Elected

1842 ACTON, WILLIAM, 17, Queen Anne street, Cavendish square. *Trans.* 1.

1851 ADAMS, JOHN, Surgeon to the London Hospital; 10, Finsbury Circus. *Trans.* 3.

1852 ADAMS, WILLIAM, Surgeon to the Royal Orthopædic Hospital; 5, Henrietta street, Cavendish square. *Trans.* 2.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde Park.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1826 ALDERSON, JAMES, M.D., F.R.S. President of the Royal College of Physicians; 17, Berkeley square. S. 1829. C. 1848. T. 1849. V.P. 1852-3. P. 1865-6. *Trans.* 3.

1843 ALDIS, CHARLES JAMES BERRIDGE, M.D., Medical Officer of Health for St. George's, Hanover square; Senior Physician to the Surrey Dispensary; and Physician to the St. Paul and St. Barnabas Dispensary; 45B, Chester square. *Trans.* 2.

1850 ALEXANDER, CHARLES REVANS, Surgeon to the Royal Infirmary for Diseases of the Eye; 6, Cork street, Bond street.

1866 ALLBUTT, THOMAS CLIFFORD, M.A. and B.M. Camb., F.L.S., Lecturer on the Practice of Physic at the Leeds School of Medicine, and Physician to the Leeds General Infirmary; 38, Park square, Leeds. *Trans.* 3.

1863 ALTHAUS, JULIUS, M.D., 18, Bryanston street, Portman square.

1862 ANDREW, EDWYN, M.D., Windsor House, Shrewsbury.

1862 ANDREW, JAMES, M.D., Assistant-Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 59, Russell square.

1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

Elected

1867 ANSTIE, FRANCIS EDMUND, M.D., Senior Assistant-Physician and Lecturer on Materia Medica at the Westminster Hospital; 16, Wimpole street, Cavendish square.

1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Trans.* 8.

1817 †ASHBURNER, JOHN, M.D., F.L.S., 1, Hyde park place, Cumberland gate. C. 1821, 1830-31.

1851 ASHTON, THOMAS JOHN, Consulting Surgeon to the St. Marylebone Infirmary; 31, Cavendish square.

1820 *BADLEY, JOHN, Dudley, Worcestershire.

1836 BAIRD, ANDREW WOOD, M.D., Physician to the Dover Hospital; 7, Camden crescent, Dover, Kent.

1851 *BAKER, ALFRED, Surgeon to the Birmingham General Hospital, and Lecturer on Surgery at Sydenham College; 20A, Temple row, Birmingham.

1865 BAKER, WILLIAM MORRANT, Demonstrator of Anatomy and Operative Surgery, and Warden of the College, St. Bartholomew's Hospital. *Trans.* 2.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Deputy Inspector-General of Hospitals; 6, Whitehall yard. C. 1852-3. V.P. 1860-1. *Trans.* 2.

1848 BALLARD, EDWARD, M.D., Medical Officer of Health for Islington; 7, Compton terrace, Upper street, Islington. *Trans.* 5.

1849 BALLARD, THOMAS, M.D., 10, Southwick place, Hyde park square.

1866 *BANKS, JOHN THOMAS, M.D., King's Professor of Physic, Physician to Richmond, Whitworth, and Hardwicke Hospitals, and Sir Patrick Dun's Hospital; Consulting Physician to the Coombe Hospital; 10, Merrion square east, Dublin.

1847 BARCLAY, ANDREW WHYTE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; Medical Officer of Health for Chelsea; 23A, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. C. 1865-6. *Trans.* 2.

1862 BARKER, EDGAR, jun., 6, Upper Hyde park street.

Elected

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Trans.* 6.

1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. Thomas's Hospital, and Physician to the Royal Maternity Charity; 46, Finsbury square. *Trans.* 3.

1864 BARRATT, JOSEPH GILLMAN, M.D.; 8, Cleveland gardens, Bayswater.

1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Clifton House, Ryde, Isle of Wight.

1859 BARWELL, RICHARD, Surgeon to, and Lecturer on Anatomy at, the Charing Cross Hospital; 32, George street, Hanover square. *Trans.* 1.

1844 BASHAM, WILLIAM RICHARD, M.D., Senior Physician to, and Lecturer on Medicine at, the Westminster Hospital; 17, Chester street, Belgrave square. S. 1852-4. C. 1860-1. V.P. 1864-5. *Trans.* 2.

1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Assistant-Physician to University College Hospital; 20, Queen Anne street. *Trans.* 1.

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of Physiology and General and Morbid Anatomy in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. *Trans.* 1.

1860 BEALEY, ADAM, M.D., M.A. Camb., Birch Lea, Harrogate.

1841 BEAMAN, GEORGE, M.D., 3, Henrietta street, Covent garden.

1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange, Newton in Cartmel, Lancashire.

1865 BEATTIE, HENRY, M.D., 15, Percy circus, Pentonville.

1836 BEAUMONT, WILLIAM RAWLINGS, Consulting Surgeon to the Toronto General Hospital, late Professor of Surgery in the University of King's College; Toronto, Canada West. *Trans.* 3.

Elected

1840 BEEVOR, CHARLES, 129, Harley street, Cavendish square.

1858 BEGLEY, WILLIAM CHAPMAN, M.D., Middlesex County Lunatic Asylum, Hanwell.

1819 †BELL, THOMAS, F.R.S., F.L.S., The Wakes, Selborne, Hants. C. 1832-3. V.P. 1854. *Trans.* 1.

1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and Mentone.

1845 BERRY, EDWARD UNWIN, 76, Gower street, Bedford square.

1820 BERTIN, STEPHEN, Paris.

1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Royal Infirmary, Liverpool, and Lecturer on Clinical Surgery in the Liverpool School of Medicine; 2, Rodney street, Liverpool.

1815 †BILLING, ARCHIBALD, M.D., F.R.S., Member of the Senate of the University of London; 6, Grosvenor gate. C. 1825. V.P. 1828-9.

1827 BIRCH, WILLIAM, Barton-under-Needwood, Staffordshire. *Trans.* 2.

1854 BIRD, PETER HINCKES, F.L.S., 1, Norfolk square, Hyde park.

1856 BIRD, WILLIAM, Surgeon to the West London Hospital; Bute House, Hammersmith.

1849 BIRKETT, EDMUND LLOYD, M.D., Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6

1851 BIRKETT, JOHN, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 59, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. *Trans.* 6. *Sci. Com.*

1866 BISHOP, EDWARD, M.D., late Lecturer on Materia Medica and Therapeutics at the Leeds School of Medicine; Cintra park, Upper Norwood.

1843 BLACK, PATRICK, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Queen Anne street, Cavendish square. C. 1856. V.P. 1866.

1847 BLACKMAN, GEORGE C., M.D., Professor of Surgery in the Medical College of Ohio; New York, U.S.

Elected

1840 BLAKISTON, PEYTON, M.D., F.R.S., St. Leonard's-on-Sea.

1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada East.

1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 3, Clarges street, Piccadilly.

1867 BLOXAM, JOHN ASTLEY, Regent's park barracks, Albany street.

1823 BOJANUS, LOUIS HENRY, M.D., Wilna.

1846 BOSTOCK, JOHN ASHTON, Hon. Surgeon to H.M. the Queen; Surgeon-Major Scots Fusilier Guards; 54, Chester square, Belgravia. C. 1861-2. *Sci. Com.*

1863 BOWEN, FRANCIS, M.D., 62, Upper Berkeley street, Portman square.

1841 BOWMAN, WILLIAM, F.R.S., F.L.S., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Trans.* 3.

1862 BRACE, WILLIAM HENRY, M.D., 1, Gay street, Bath.

1867 *BRETT, ALFRED T., M.D., Lime-tree House, Watford, Herts.

1867 BRIDGWATER, THOMAS, M.B. Lond., Harrow-on-the-Hill, Middlesex.

1868 BROADBENT, WILLIAM HENRY, M.D., Assistant-Physician to, and Lecturer on Physiology at, St. Mary's Hospital; Assistant-Physician to the London Fever Hospital; 44, Seymour street, Portman square.

1851 BRODHURST, BERNARD EDWARD, F.L.S., Assistant-Surgeon to St. George's Hospital, and to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868. *Trans.* 2. *Pro.* 1.

1844 †BROOKE, CHARLES, M.A., F.R.S., *Librarian*, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 16, Fitzroy square. C. 1855. L. 1866-68.

1854 *BROWN, HENRY, Windsor, Berks.

1857 *BROWN, ROBERT, Surgeon to the Cumberland Dispensary; 5, Devonshire street, Carlisle.

Elected

1860 BROWN-SEQUARD, CHARLES EDOUARD, M.D., F.R.S., Professor of Physiology and Pathology, Massachusetts Medical College, Harvard University, Boston, U.S.; Paris. *Sci. Com.*

1851 BROWNE, ALEXANDER, M.D., Twynholm, Kirkcudbright.

1867 BRUCE, ALEXANDER, Assistant-Surgeon to, and Lecturer on Anatomy at, the Westminster Hospital; 8, Old Cavendish street, Cavendish square.

1867 BRUNJES, MARTIN, 42, Brook street, Grosvenor square.

1860 BRYANT, THOMAS, Assistant-Surgeon to, and Demonstrator of Operative Surgery at, Guy's Hospital; 2, Finsbury Square. *Trans.* 7; *Pro.* 1. *Sci. Com.*

1855 BRYANT, WALTER JOHN, L.R.C.P. Edinb.; 23A, Sussex square, Hyde park gardens.

1823 BUCHANAN, B. BARTLET, M.D.

1864 BUCHANAN, GEORGE, M.D., Physician to the London Fever Hospital, and Assistant-Physician to the Hospital for Sick Children; Medical Inspector for the Privy Council; Medical Officer of Health for St. Giles District; 53, Harley street, Cavendish square.

1864 BUCKLE, FLEETWOOD, M.D., H.M.S. 'Victory,' Portsmouth.

1839 BUDD, GEORGE, M.D., F.R.S., Consulting Physician to the Seamen's Hospital Ship 'Dreadnought;' Needham, Suffolk. C. 1846-7. V.P. 1857. *Trans.* 5.

1833 †BURROWS, GEORGE, M.D., F.R.S., President of the Medical Council; Consulting Physician to St. Bartholomew's Hospital; Physician to Christ's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V.P. 1849-50. *Trans.* 2.

1820 BURROWS, SAMUEL.

1837 †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital Ship 'Dreadnought;' 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Trans.* 4.

1818 BUTTER, JOHN, M.D., F.R.S., F.L.S., Physician Extraordinary to the Plymouth Royal Eye Infirmary; Windsor villa, Plymouth.

Elected

1868 BUZZARD, THOMAS, M.D., Assistant-Physician to the National Hospital for the Epileptic and Paralysed; 12, Green street, Grosvenor square.

1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.

1851 CALLAWAY, THOMAS, Maison Limozin, Place Bresson, Algiers.

1861 CALLENDER, GEORGE WILLIAM, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 2. *Sci. Com.*

1852 *CANNEY, GEORGE, M.D., Bishop-Auckland, Darlington, Durham.

1847 CARLILL, JOHN BURFORD, M.D., Surgeon-Accoucheur to the Newman street Lying-in Institution; 42, Weymouth street, Portland place.

1853 CARTER, ROBERT BRUDENELL, Stroud, Gloucestershire.

1845 CARTWRIGHT, SAMUEL, Professor of Dental Surgery at King's College, London; Surgeon-Dentist to King's College Hospital; 32, Old Burlington street. C. 1860–1. *Sci. Com.*

1839 CATHROW, WILLIAM. C. 1860-1.

1868 CAVAFY, JOHN, M.B. Lond., Lecturer on Morbid Anatomy at the Westminster Hospital; 5, Whitehall.

1845 CHALK, WILLIAM OLIVER, Surgeon to the St. Marylebone Eye and Ear Institution; 3, Nottingham terrace, York gate, Regent's park.

1844 CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Lock Hospital; 64, Brook street, Grosvenor square. *Trans.* 1. C. 1861. V.P. 1867.

1859 CHANCE, FRANK, M.D., Croft Lodge, Cambridge.

1849 CHAPMAN, FREDERICK, Old Friars, Richmond green, Surrey.

1837 †CHAPMAN, HENRY THOMAS, 21, Lower Seymour street, Portman square. C. 1858.

Elected

1868 CHEADLE, WALTER BUTLER, M.D., Assistant-Physician to St. Mary's Hospital; 2, Hyde park place, Cumberland gate.

1852 CHILDS, GEORGE BORLASE, Surgeon-in-Chief to the City Police Force; 11, Finsbury place south.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital; 40, Russell square.

1842 CHOWNE, WILLIAM DINGLE, M.D., Consulting Physician to the Charing Cross Hospital; Corresponding Fellow of the Royal Academy of Surgery of Madrid; 17, Hyde park place, Cumberland gate. C. 1853-4.

1866 CHURCH, WILLIAM SELBY, M.D., Reader in Anatomy, Christ Church, Oxford; Assistant-Physician to, and Demonstrator of Morbid Anatomy at, St. Bartholomew's Hospital; Granville chambers, Granville street, Portman square.

1860 CLARK, ANDREW, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 23, Montague place, Russell square.

1839 †CLARK, FREDERICK LE GROS, Professor of Human Anatomy and Surgery to the Royal College of Surgeons; Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Surgeon to the Magdalen Hospital, and Consulting Surgeon to the Royal Surrey County Hospital; Examiner in Surgery to the University of London; 14, St. Thomas's street, Southwark, and Lee, Kent. S. 1847-9. V.P., 1855-6. *Trans.* 3.

1848 CLARKE, JOHN, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; Physician to the General Lying-in Hospital; 42, Hertford street, May fair. C. 1866.

1866 CLARKE, WILLIAM FAIRLIE, Surgeon to the St. George's and St. James's Dispensary; 1, Curzon street, May fair.

1861 *CLARKE, WILLIAM JAMES, Surgeon to the Huddersfield Infirmary; John-William street, Huddersfield, Yorkshire.

Elected

1850 CLARKSON, JOSIAH, New Hall street, Birmingham. *Trans.* 1.

1842 CLAYTON, OSCAR MOORE PASSEY, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street. C. 1865.

1853 CLOVER, JOSEPH THOMAS, 3, Cavendish place, Cavendish sq.

1857 COATES, CHARLES, F.R.C.P. Edinb., Physician to the Bath United General Hospital; 10, Circus, Bath.

1851 COCK, EDWARD, Senior Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; Consulting Surgeon to the Asylum for Deaf and Dumb; Dean street south, Tooley street, Southwark. C. 1857. *Trans.* 3.

1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 13, Brook street, Hanover square. *Trans.* 2.

1850 COHEN, DANIEL WHITAKER, M.D., South Bank, North Down lane, Bideford, Devon.

1818 COLE, ROBERT, F.L.S., Holybourne, Hampshire.

1854 COLLINS, FREDERICK, M.D., Medical Officer of Health for Wanstead; Wanstead lodge, Essex.

1867 COOKE, T. C. WEEDEN, Senior Surgeon to the Royal Free Hospital, and to the Cancer Hospital; 76, Upper Berkeley street, Portman square.

1840 *COOKE, WILLIAM ROBERT, 2, Carlton villas, Hencroft street, Slough.

1865 COOPER, ALFRED, Assistant-Surgeon to St. Mark's Hospital; Junior Surgeon to the West London Hospital; 70, Jermyn street, Piccadilly.

1819 COOPER, GEORGE, Brentford, Middlesex.

1841 COOPER, GEORGE LEWIS, one of the Surgeons to the National Vaccine Institution, and Teacher of Vaccination to the Medical School of University College; Surgeon to the Bloomsbury Dispensary; 7, Woburn place, Russell square. C. 1860-1. *Trans.* 1.

1843 COOPER, WILLIAM WHITE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9.

Elected

1841 COOTE, HOLMES, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 7, Princes street, Hanover square. S. 1853-4. C. 1864-5. *Trans.* 2.

1835 COPELAND, GEORGE FORD, Cheltenham.

1822 †COPLAND, JAMES, M.D., F.R.S., Consulting Physician to the Royal Infirmary for Children, and to the Great Northern Hospital; Hon. Fellow of the Royal Academy of Sciences of Sweden, &c.; 5, Old Burlington street. C. 1831. V.P. 1838-9. P. 1853-4.

1851 CORBET, JOHN. *Trans.* 2.

1868 CORNISH, WILLIAM ROBERT, Surgeon, Madras Army; Secretary to the Inspector-General, Indian Medical Department; [95, Park street, Grosvenor square.]

1860 *CORRY, THOMAS CHARLES STEUART, M.D., Surgeon to the Belfast General Dispensary; 9, Clarendon place, Belfast.

1839 *CORSELLIS, CHARLES CÆSAR, M.D., F.L.S., Benson, Oxon.

1853 CORY, WILLIAM GILLETT, M.D.

1847 COTTON, RICHARD PAYNE, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; 46, Clarges street, Piccadilly. C. 1863.

1828 †COULSON, WILLIAM, F.L.S., Consulting Surgeon to St. Mary's Hospital, and to the German Hospital; 2, Frederick's place, Old Jewry, and 1, Chester terrace, Regent's park. C. 1831. L. 1832-7. V.P. 1851-2. *Trans.* 1.

1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 29, St. James's place.

1860 †COUPER, JOHN, Surgeon to the Out-Patients at the London Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital; 28, Park street, Grosvenor square.

1862 COWELL, GEORGE, Surgeon to the St. George's and St. James's Dispensary; 65, Belgrave road, Pimlico.

1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Wiesbaden. C. 1853-4.

1868 CRAWFORD, THOMAS, M.D., 6, Whitehall yard.

Elected

1847 CRITCHETT, GEORGE, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 21, Harley street, Cavendish square. C. 1865. *Trans.* 1.

1868 CROFT, JOHN, Assistant-Surgeon to St. Thomas's Hospital; the Hospital.

1862 CROMPTON, SAMUEL, 17A, Princess street, Manchester.

1837 CROOKES, JOHN FARRAR, 5, Waterloo crescent, Dover.

1860 CROSS, RICHARD, M.D., 5, Queen street, Scarborough.

1849 *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.

1851 CUMMING, JAMES CAMERON, M.D., 1, Cadogan place, Sloane street.

1865 CURGENVEN, J. BRENDON, 11, Craven hill gardens, Bayswater.

1846 CURLING, HENRY, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.

1837 †CURLING, THOMAS BLIZARD, F.R.S., Surgeon to the London Hospital; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. *Trans.* 13. *Pro.* 1. *Sci. Com.*

1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.

1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.

1852 CUTLER, THOMAS, M.D., Acting Physician at the Spa Waters; Spa, Belgium.

1836 *DANIEL, JAMES STOCK, Ramsgate, Kent.

1848 DAUBENY, HENRY, San Remo, Italy.

1846 DAVIES, FREDERICK, M.D., 124, Gower street, Bedford sq.

1847 DAVIES, JOHN, M.D., Physician-Extraordinary to the Hertford General Infirmary, and Visiting Physician to the Hadham Palace Lunatic Asylum, Hertford.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 10, Gay street, Bath.

1852 DAVIS, JOHN HALL, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Physician to the Royal Maternity Charity, and Consulting Physician-Accoucheur to the St. Pancras Infirmary; 24, Harley street, Cavendish square.

1818 DAWSON, JAMES, Wray Castle, Windermere.

Elected

1847 DAY, GEORGE EDWARD, M.D., F.R.S., Emeritus Professor of Medicine in the University of St. Andrews; Furzewell House, Torquay.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Margaret street Dispensary for Consumption; 10, Manchester square.

1867 DE MERIC, VICTOR, Surgeon to the Royal Free Hospital, and to the German Hospital, Dalston; 52, Brook street, Grosvenor square.

1846 *DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.

1859 DICKINSON, WILLIAM HOWSHIP, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Assistant-Physician to the Hospital for Sick Children; 11, Chesterfield street, May fair. *Trans.* 8. *Sci. Com.*

1844 DICKSON, ROBERT, M.D., F.L.S. C. 1860.

1839 †DIXON, JAMES, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Consulting Ophthalmic Surgeon to the Asylum for Idiots; 2, Portman square. L. 1849-55. V.P., 1857-8. T. 1863-4. C. 1866-7. *Trans.* 4.

1862 DOBELL, HORACE B., M.D., Physician to the Royal Infirmary for Diseases of the Chest, City road; 84, Harley street. *Trans.* 1.

1845 DODD, JOHN.

1857 DOUGLAS, ARCHIBALD, M.D., 8, Clifton place, Sussex square, Hyde park.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to, and Lecturer on Materia Medica and Therapeutics at, the London Hospital; 39, Welbeck street, Cavendish square. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1853 DRUITT, ROBERT, M.R.C.P., 37, Hertford street, May fair. *Trans.* 2.

1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 99, Southampton row, Russell square.

Elected

1865 DUCKWORTH, DYCE, M.D., Medical Tutor to St. Bartholomew's Hospital; Physician to the Royal General Dispensary; 70, Wimpole street.

1845 DUFF, GEORGE, M.D., High street, Elgin.

1845 DUFFIN, EDWARD WILLSON, 18, Devonshire street, Portland place. *Trans.* 1.

1867 DUKES, M. CHARLES, M.D., High House, Enfield Highway.

1833 †DUNN, ROBERT, 31, Norfolk street, Strand. C. 1845-6. *Trans.* 2.

1861 DU PASQUIER, CLAUDIUS FRANCIS, Surgeon-Apothecary to H.M. the Queen, and to the Household of H.R.H. the Prince of Wales; 62, Pall Mall.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 82, Brook street, Grosvenor square. *Trans.* 2. *Sci. Com.*

1843 DURRANT, CHRISTOPHER MERCER, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839 DYER, HENRY SUMNER, M.D., Sennowe Hall, Guist, Norfolk. C. 1854-5.

1836 EARLE, JAMES WILLIAM, late of Norwich.

1868 EASTES, GEORGE, M.B. Lond., 43, Trinity square, Southwark.

1824 EDWARDS, GEORGE.

1823 EGERTON, CHARLES CHANDLER, Kendall Lodge, Epping.

1861 *ELLIOT, ROBERT, M.D., Physician to the Carlisle Dispensary; 35, Lowther street, Carlisle.

1848 ELLIS, GEORGE VINER, Examiner in Anatomy at the University of London; Professor of Anatomy in University College, London. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., Medical Superintendent, St. Luke's Hospital, Old street.

1854 *ELLISON, JAMES, M.D., Surgeon in Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1835 ENGLAND, WILLIAM, M.D., Ipswich, Suffolk.

1842 ERICHSEN, JOHN E., *Vice-President*, Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Examiner in Surgery at the University of London; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. *Trans.* 2.

Elected

1836 EVANS, GEORGE FABIAN, M.D., late Physician to the General Hospital, Birmingham.

1815 *EVANS, GRIFFITH FRANCIS DORSETT, M.D.; Crescent, Shrewsbury. C. 1838.

1845 EVANS, WILLIAM JULIAN, M.D., Pinner, Middlesex.

1864 FAGGE, CHARLES HILTON, M.D., Assistant-Physician to Guy's Hospital; Physician to the Royal Infirmary for Diseases of Children and Women, Waterloo road; 12, Union street, Southwark.

1858 FALCONER, RANDLE WILBRAHAM, M.D., Physician to the Bath United Hospital; 22, Bennett street, Bath.

1862 FARQUHARSON, ROBERT, M.D., Medical Officer, Rugby School.

1844 FARRE, ARTHUR, M.D., F.R.S., Physician-Accoucheur to H.R.H. the Princess of Wales and H.R.H. the Princess Louis of Hesse; 12, Hertford street, May fair. C. 1857. V.P. 1864. *Sci. Com.*

1863 FENWICK, SAMUEL, M.D., Assistant-Physician to the London Hospital; 29, Harley street, Cavendish square. *Trans.* 3.

1841 FERGUSSON, SIR WILLIAM, Bart., F.R.S., Sergeant-Surgeon to H.M. the Queen; Professor of Surgery in King's College, London, and Surgeon to King's College Hospital; 16, George street, Hanover square. C. 1849-50. V.P. 1863-4. *Trans.* 4.

1852 *FIELD, ALFRED GEORGE, Surgeon to St. Mary's Hospital, Brighton; 28, Old Steine, Brighton.

1849 FINCHAM, GEORGE TUPMAN, M.D., Physician to, and Lecturer on Clinical Medicine at, the Westminster Hospital; 13, Belgrave road.

1866 FISH, JOHN CROCKETT, B.A., M.B. Camb., 10, Vere street, Oxford street.

1836 †FISHER, SIR JOHN WILLIAM, 5, Grosvenor gate. C. 1843-4.

1860 FITZGERALD, THOMAS GEORGE, Staff-Surgeon; 6, Whitehall yard.

1866 FITZPATRICK, THOMAS, M.D., M.A., Dublin; 30, Sussex gardens, Hyde park.

Elected

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Physician to the Birmingham General Hospital; Waterloo street, Birmingham. *Trans.* 1.

1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Hanley, Stoke-on-Trent, Staffordshire.

1848 FORBES, JOHN GREGORY, Surgeon to the Metropolitan Convalescent Institution; 82, Oxford terrace, Hyde park. C. 1868. *Trans.* 2.

1852 †FORSTER, JOHN COOPER, Assistant-Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Surgeon to the Royal Infirmary for Children; 10, St. Thomas's street, Southwark. C. 1868. *Pro.* 1.

1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Anatomy at the Queen's College, Birmingham, and Assistant-Physician to the Queen's Hospital; 4, Old Square, Birmingham, and Grosvenor house, Edgbaston.

1859 FOX, EDWARD LONG, M.B., Physician to the Bristol Royal Infirmary; 1, Chesterfield place, Clifton.

1858 *FOX, WILSON, M.D., Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 22B, Cavendish Square. *Trans.* 2.

1841 FRANZ, JOHN CHRISTOPHER AUGUSTUS, M.D.

1843 FRASER, PATRICK, M.D. C. 1866.

1868 FREEMAN, WILLIAM HENRY, 29, Spring Gardens.

1836 †FRENCH, JOHN GEORGE, Surgeon to the St. James's Infirmary; 41, Great Marlborough street. C. 1852-3.

1849 FRERE, ROBERT TEMPLE, M.A., F.R.C.P., 143, Harley street.

1846 FULLER, HENRY WILLIAM, M.D., *Vice-President*, Physician to St. George's Hospital; 13, Manchester square. C. 1862. S. 1864-5. V.P. 1868. *Trans.* 3.

1864 *GAIRDNER, WILLIAM TENNANT, M.D., Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 21, Blythswood square, Glasgow.

1865 GANT, FREDERICK JAMES, Surgeon and Pathological Anatomist to the Royal Free Hospital; 16, Connaught square, Hyde Park.

Elected

1867 GARLAND, EDWARD CHARLES, L.R.C.P. Ed., Yeovil, Somerset.

1867 GARLIKE, THOMAS W., Tulse Hill, Brixton.

1854 GARROD, ALFRED BARING, M.D., F.R.S., Professor of Materia Medica in King's College, London, and Physician to King's College Hospital; Examiner in Materia Medica at the University of London; 11, Harley street, Cavendish square. C. 1867. *Trans.* 8.

1857 GASCOYEN, GEORGE GREEN, *Secretary*, Surgeon to the Lock Hospital; Assistant-Surgeon to, and Lecturer on Anatomy at, St. Mary's Hospital; 48, Queen Anne street, Cavendish square. S. 1866-68. *Trans.* 2. *Sci. Com.*

1851 GASKOIN, GEORGE, 7, Westbourne park.

1819 GAULTER, HENRY.

1848 GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south.

1866 GEE, SAMUEL JONES, M.D., Assistant-Physician to St. Bartholomew's Hospital; Assistant-Physician to the Hospital for Sick Children; 46, Queen Anne street, Cavendish square.

1821 *GEORGE, RICHARD FRANCIS, 20, Marlborough buildings, Bath.

1864 GIBB, SIR GEORGE DUNCAN, Bart., M.D., LL.D., Assistant-Physician to, and Lecturer on Forensic Medicine at, the Westminster Hospital; 1, Bryanston street, Portman square. *Trans.* 1.

1858 GODFREY, BENJAMIN, M.D., Carlton House, Enfield, Middlesex.

1867 GOODEVE, EDWARD, M.B., Hon. Physician to H.M. the Queen; late Surgeon-Major, H.M.'s Bengal army; 40, Park street, Grosvenor square.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Physician to the Middlesex Hospital; 5, Savile row, Burlington gardens, C. 1864-5. *Trans.* 2.

1818 GORDON, JAMES ALEXANDER, M.D., C.B., F.R.S., Pixholme, near Dorking, Surrey. C. 1828. V.P. 1829. *Trans.* 1.

Elected

1862 GOULSTON, JOHN G., M.D., 30, Clarence street, Liverpool.

1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; 34, Finsbury square.

1844 GRANTHAM, JOHN, Crayford, Kent.

1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; 2, Upper Brook street, Grosvenor square. C. 1863.

1868 GREEN, THOMAS HENRY, M.D., Lecturer on Pathology at Charing Cross Hospital; 74, Wimpole street.

1843 GREENHALGH, ROBERT, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital; Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 72, Grosvenor street.

1860 GREENHOW, EDWARD HEADLAM, M.D., Examiner in Forensic Medicine at the University of London; Assistant-Physician to, and Lecturer on Public Health and on Medical Jurisprudence at, the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; 77, Upper Berkeley street, Portman square. *Trans.* 2.

1868 GRIGG, WILLIAM CHAPMAN, 1, Windsor terrace, Maida Hill.

1814 GROVE, JOHN, M.D., Salisbury.

1852 GROVE, JOHN, West Hill, Wandsworth, Surrey.

1860 GUENEAU DE MUSSY, HENRI, M.D., Physician to the French Hospital, Lisle street, Leicester Square; 4, Cavendish place, Cavendish square.

1849 GULL, WILLIAM WITHEY, M.D., D.C.L., Member of the Senate of the University of London; 74, Brook street, Grosvenor square. C. 1864. *Trans.* 2.

1837 GULLY, JAMES MANBY, M.D., Great Malvern, Worcestershire.

1854 HABERSHON, SAMUEL OSBORNE, M.D., Physician to, and Lecturer on Materia Medica and Therapeutics at, Guy's Hospital; Examiner in Materia Medica at the University of London; 70, Brook street, Grosvenor square. S. 1867. *Trans.* 3.

Elected

1849 HAILEY, HAMMETT, F.L.S., Newport Pagnell, Bucks.

1848 HALLEY, ALEXANDER, M.D., F.G.S., 16, Harley street, Cavendish square.

1819 †HAMMERTON, THOMAS, 112, Piccadilly. C. 1829-30.

1838 HANCOCK, HENRY, Surgeon to the Charing Cross Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; 76, Harley street, Cavendish square. C. 1851.

1849 HANSARD, RICHARD JAMES, late Surgeon to the Radcliffe Infirmary, Oxford.

1848 *HARCOURT, GEORGE, M.D., Chertsey, Surrey.

1836 HARDING, JOHN FOSSE, Mount Sandford, Southborough, Tunbridge Wells. C. 1858-9.

1856 HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 57, Brook street, Grosvenor square.

1857 HARLEY, GEORGE, M.D., F.R.S., Professor of Medical Jurisprudence in University College, London; and Physician to University College Hospital; 25, Harley street, Cavendish square. *Trans.* 1. *Sci. Com.* 2.

1861 HARLEY, JOHN, M.D., F.L.S., Assistant-Physician to the London Fever Hospital; 78, Upper Berkeley street, Portman square. *Trans.* 2.

1866 HARPER, PHILIP H., 30, Cambridge street, Hyde park.

1859 HARRIS, FRANCIS, M.D., F.L.S., Physician to, and Lecturer on Botany at, St. Bartholomew's Hospital; Assistant-Physician to the Hospital for Sick Children; 24, Cavendish square.

1841 HARVEY, WILLIAM, Surgeon to the Royal Dispensary for Diseases of the Ear, and to the Freemasons' Female Charity; Aural Surgeon to the Great Northern Hospital; 2, Soho square. C. 1854.

1854 HAVILAND, ALFRED, late Surgeon to the Bridgewater Infirmary; (London).

1828 †HAWKINS, CÆSAR HENRY, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.

Elected

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Trans.* 2.

1848 HAWKSLEY, THOMAS, M.D., Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 6, Brook street, Hanover square.

1860 HAYWARD, HENRY HOWARD, Assistant Surgeon-Dentist to St. Mary's Hospital; Dental Surgeon to the Hospital for Consumption, Brompton; 38, Harley street, Cavendish square.

1861 HAYWARD, WILLIAM HENRY, Church House, Oldbury, near Birmingham.

1848 *HEALE, JAMES NEWTON, M.D., Physician to the Winchester County Hospital; Winchester, Hants.

1865 HEATH, CHRISTOPHER, Assistant-Surgeon to University College Hospital; and Teacher of Operative Surgery in, University College, London; 9, Cavendish place, Cavendish square.

1850 HEATON, GEORGE, M.D., Boston, U.S.

1829 †HEBERDEN, THOMAS, M.D., 98, Park street, Grosvenor square.

1844 HENNEN, JOHN, M.D. L. 1848-50.

1849 HENRIQUES, AMOS, M.D., Hon. Physician to the Spanish Embassy; 67, Upper Berkeley street, Portman square.

1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.

1843 HEWETT, PRESCOTT GARDNER, Surgeon-Extraordinary to H.M. the Queen; Surgeon to St. George's Hospital; 1, Chesterfield street, May fair. C. 1859. V.P. 1866-7. *Trans.* 7. *Sci. Com.*

1855 HEWITT, GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; 36, Berkeley square.

1853 HEWLETT, THOMAS, Surgeon to Harrow School; Harrow, Middlesex. *Trans.* 1.

1868 HILL, JOHN D., Surgeon to the Royal Free Hospital; 17, Guilford street, Russell square.

Elected

1862 HILL, MATTHEW BERKELEY, M.B. Lond., Assistant-Surgeon to University College Hospital; Surgeon to the Lock Hospital; 14, Weymouth street, Portland place.

1867 HILL, SAMUEL, M.D., 22, Mecklenburgh square.

1854 HILLIER, THOMAS, M.D., Physician to the Hospital for Sick Children; Physician to the Skin Department of University College Hospital; and Medical Officer of Health for St. Pancras; 32, Queen Anne street, Cavendish square. *Trans.* 2.

1842 HILLMAN, WILLIAM AUGUSTUS, Senior Assistant-Surgeon to the Westminster Hospital; 1, Argyll street, Regent street. C. 1858-9.

1841 †HILTON, JOHN, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Surgeon to Guy's Hospital; Consulting Surgeon to the Royal General Dispensary, St. Pancras; 10, New Broad street, City. C. 1851. V.P. 1863-4. *Trans.* 3.

1868 HINTON, JAMES, Aural Surgeon to Guy's Hospital; 18, Savile Row. *Trans.* 2.

1859 HIRD, FRANCIS, Surgeon to, and Lecturer at, the Charing Cross Hospital; 13, Old Burlington street.

1813 HODGSON, JOSEPH, F.R.S., 60, Westbourne terrace, Hyde park gardens. C. 1817. P. 1851-2. *Trans.* 1.

1861 *HOFFMEISTER, WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Cowes, Isle of Wight.

1843 HOLDEN, LUTHER, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Dispensary; Surgeon to the Foundling Hospital; 65, Gower street, Bedford square. C. 1859. L. 1865.

1814 †HOLLAND, SIR HENRY, Bart., M.D., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen; 72, Brook street, Grosvenor square. C. 1817, 1833-4. V.P. 1826, 1840. *Trans.* 1.

1868 HOLLIS, WILLIAM AINSLIE, M.A., M.B. Camb., 15, Conduit street, Regent street.

1861 HOLMAN, WILLIAM HENRY, M.B. Lond., 68, Adelaide road south, Hampstead.

Elected

1856 HOLMES, TIMOTHY, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; Surgeon in Chief to the Metropolitan Police Force; 31, Clarges street, Piccadilly. *Trans.* 4. *Sci. Com.*

1846 HOLT, BARNARD WIGHT, Senior Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3.

1846 HOLTHOUSE, CARSTEN, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the South London Ophthalmic Hospital; 2, Storey's gate, St. James's Park. C. 1863.

1853 HOOD, SIR WILLIAM CHARLES, M.D., F.L.S., Visiting Physician in Lunacy to the Court of Chancery; Croydon Lodge, Surrey. *Trans.* 1.

1865 HOWARD, BENJAMIN, M.D., Lecturer on Operative Surgery in the Medical College of the University of New York; 327, West 23rd street, New York.

1865 HOWARD, EDWARD, M.D., Redhill, Surrey.

1868 HUBBARD, JOHN WADDINGTON, 16, Pembroke gardens, Kensington.

1857 HULKE, JOHN WHITAKER, F.R.S., Assistant-Surgeon to, and Lecturer on Physiology and Ophthalmic Surgery at, the Middlesex Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Lecturer on Anatomy and Physiology to the Royal College of Surgeons; 10, Old Burlington street. *Trans.* 4. *Sci. Com.*

1857 HULME, EDWARD CHARLES, Surgeon to the Great Northern Hospital; Surgeon to the Central London Ophthalmic Hospital; 38, Gower street, Bedford square. *Trans.* 1.

1844 HUMBY, EDWIN, 83, Hamilton terrace, St. John's wood. C. 1866-7.

1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Human Anatomy and Physiology in the Cambridge University Medical School; Cambridge. *Trans.* 4.

1866 HUNTER, CHARLES, 30, Wilton place, Belgrave square.

Elected

1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 104, St. Aldate's, Oxford. *Trans.* 1.

1856 HUTCHINSON, JONATHAN, Surgeon to, and Lecturer on Surgery at, the London Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 4, Finsbury Circus. *Trans.* 1 *Pro.* 2.

1820 HUTCHINSON, WILLIAM, M.D.

1840 HUTTON, CHARLES, M.D., Senior Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1866 ILES, FRANCIS HENRY WILSON, M.D., Watford, Herts.

1847 IMAGE, WILLIAM EDMUND, Senior Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D., 10, New Cavendish Street, Portland place.

1841 †JACKSON, PAUL, 24, Wimpole street, Cavendish square. C. 1862.

1868 JACKSON, THOMAS CARR, Surgeon to the Great Northern Hospital, and Surgeon to the National Orthopædic Hospital; 3, Weymouth street, Portland place.

1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital; Darlington street, Wolverhampton.

1841 JACOBOVICS, MAXIMILIAN MORITZ, M.D., Vienna.

1825 JAMES, JOHN B., M.D.

1844 JEAFFRESON, SAMUEL JOHN, M.D., Consulting Physician to the Warneford Hospital and Warwick Dispensary; Leamington, Warwickshire.

1839 JEFFREYS, JULIUS, F.R.S., Drymona, Belvidere road south, Upper Norwood, Surrey.

1840 *JENKS, GEORGE SAMUEL, M.D., 18, Circus, Bath.

1851 JENNER, SIR WILLIAM, Bart., M.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1864. *Trans.* 3.

Elected

1848 JOHNSON, ATHOL ARCHIBALD WOOD, 20, Regency square, Brighton. *Trans.* 1.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Genevois."

1847 JOHNSON, GEORGE, M.D., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. *Trans.* 8.

1862 JONES, CHARLES HANDFIELD, M.B., F.R.S., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; 49, Green street, Grosvenor square.

1844 †JONES, HENRY BENCE, M.A., M.D., F.R.S., 84, Brook street, Grosvenor square. C. 1855-6. V.P. 1866. *Trans.* 11.

1835 †JONES, HENRY DERVICHE, 12, Norfolk crescent, Oxford square. C. 1854-5.

1837 †JONES, THOMAS WILLIAM, M.D., 55, St. John's Park, Upper Holloway, and 3, North buildings, Finsbury. C. 1858.

1859 JONES, WILLIAM PRICE, M.D., Surbiton, Kingston.

1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Senior Surgeon to the Birmingham Eye and Ear Hospital; Medical Inspector of Factories; 16, Colmore row, Birmingham.

1816 *KAUFFMAN, GEORGE HERMANN, M.D., Hanover.

1848 *KENDELL, DANIEL BURTON, M.D., Kettlethorpe Hall, Wakefield, Yorkshire.

1847 KEYSER, ALFRED, 21, Norfolk crescent, Oxford square.

1857 KIALLMARK, HENRY WALTER, 66, Prince's square, Bayswater.

1851 KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, City. C. 1866-7. *Trans.* 1. *Sci. Com.*

1855 LANE, JAMES ROBERT, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; Surgeon to the Lock Hospital; 2, Berkeley st., Piccadilly. *Trans.* 1.

Elected

1840 LANE, SAMUEL ARMSTRONG, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital; Consulting Surgeon to the Lock Hospital; 2, Berkeley street, Piccadilly. C. 1849-50. V.P. 1865.

1865 LANGTON, JOHN, Assistant-Surgeon to, and Demonstrator of Anatomy at, St. Bartholomew's Hospital; Assistant-Surgeon to the City of London Truss Society; the College, St. Bartholomew's Hospital.

1841 *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.

1862 LATHAM, PETER WALLWORK, M.A., M.B., Physician to Addenbrooke's Hospital, Cambridge; Examiner for Medical Degrees in Cambridge University; 15, Sidney street, Cambridge.

1816 LAWRENCE, G. E.

1840 LAYCOCK, THOMAS, M.D., F.R.S.E., Professor of the Practice of Medicine and of Clinical Medicine, and Lecturer on Psychology and Mental Diseases, in the University of Edinburgh; Physician to the Edinburgh Royal Infirmary; 4, Rutland street, Edinburgh.

1843 *LEACH, JESSE, Moss Hall, Heywood, near Bury, Lancashire.

1868 LEARED, ARTHUR, M.D., Senior Physician to the Great Northern Hospital; 12, Old Burlington street.

1822 LEDSAM, JOHN JOSEPH, M.D., 17, Esplanade, Scarborough, Yorkshire.

1823 †LEE, HENRY, M.D., Weather Oak, Alvechurch, near Bromsgrove, Worcestershire. C. 1837. S. 1839-40.

1843 LEE, HENRY, *Vice-President*, Surgeon to, and Lecturer on Pathology at, St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868. *Trans.* 10. *Pro.* 1. *Sci. Com.*

1822 †LEE, ROBERT, M.D., F.R.S., Corresponding Member of the Imperial Academy of Medicine, Paris; 4, Savile row, Burlington gardens. C. 1829, 1834. S. 1830-3. V.P. 1835. *Trans.* 27.

1836 LEIGHTON, FREDERICK, M.D., Frankfort-on-the-Maine.

Elected

1868 LEVY, JULIUS LAWRENCE, L.R.C.P. Ed.; 100, Westbourne terrace.

1806 LIND, JOHN, M.D.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1824 †LOCOCK, SIR CHARLES, Bart., M.D., D.C.L., F.R.S., First Physician-Accoucheur to H.M. the Queen; Member of the Senate of the University of London; 26, Hertford street, May fair. C. 1826. V.P. 1841. P. 1857-8. *Trans.* 1.

1852 LODGE, CHARLES, M.D., "United States Army."

1846 LOMAX, HENRY THOMAS, Surgeon to the County Police; St. Mary's Grove, Stafford.

1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen, Deputy Inspector-General, and Professor of Clinical and Military Surgery, New Army Medical School, Royal Victoria Hospital, Netley, Southampton. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1852 LUKE, JAMES, F.R.S., Consulting Surgeon to the London Hospital, Woolley Lodge, Maidenhead Thicket, Berks. C. 1858. *Trans.* 4.

1857 LYON, FELIX WILLIAM, M.D., Littlecote, Hungerford, Hants.

1867 MABERLY, GEORGE FREDERICK, Hillside House, Godalming, Surrey.

1867 *MAC CORMAC, WILLIAM, M.A., M.D. Dubl., 4, Howard street, Belfast.

1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 14, Lower Pembroke street, Dublin. *Trans.* 1.

1846 M'EWEN, WILLIAM, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.

1866 MACGOWAN, ALEXANDER THORBURN, Moreland House, Martock, Somersetshire.

Elected

1823 †MACILWAIN, GEORGE, Consulting Surgeon to the Finsbury Dispensary, and the St. Anne's Society's Schools; 3, Albany Court yard. C. 1829-30. V.P. 1848. *Trans.* 1.

1822 MACINTOSH, RICHARD, M.D.

1859 *M'INTYRE, JOHN, M.D., Odiham, Hants.

1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1844 MACLACHLAN, DANIEL, M.D., Deputy Inspector-General of Hospitals; Ventnor, Isle of Wight. C. 1860-1. *Trans.* 1.

1860 MACLEAN, JOHN, M.D., 24, Portman street, Portman square.

1849 MACLURE, DUNCAN MACLACHLAN, M.B., Lecturer on Physiology at the Westminster Hospital; 34, Harley street, Cavendish square.

1842 MACNAUGHT, JOHN, M.D., Bedford street, Liverpool.

1855 MARCET, WILLIAM, M.D., F.R.S., Assistant-Physician to the Brompton Hospital for Consumption; 48, Harley street. *Trans.* 2. *Sci. Com.*

1848 MARKHAM, WILLIAM ORLANDO, M.D., Poor Law Inspector for the Metropolitan District; 8, Harley street, Cavendish square. C. 1862-3. *Trans.* 2.

1867 MARSH, F. HOWARD, Assistant-Surgeon to the Hospital for Sick Children; St. Bartholomew's Hospital.

1838 MARSH, THOMAS PARR, M.D.

1851 MARSHALL, JOHN, F.R.S., Professor of Surgery in University College, London, and Surgeon to University College Hospital; 10, Savile row, Burlington gardens. C. 1866. *Trans.* 2.

1841 MARTIN, SIR JAMES RANALD, C.B., F.R.S., Examining Medical Officer to the Secretary of State for India in Council; President of Medical Board for Examination of Officers of H.M.'s Indian Medical Service; Inspector General of Hospitals; 37, Upper Brook street, Grosvenor square. C. 1853. V.P. 1862.

1853 MASFEN, WILLIAM EDWARD, Surgeon to the Staffordshire General Infirmary; Stafford.

1864 MASON, FRANCIS, Assistant-Surgeon to the Westminster Hospital; 10, Conduit street, Regent street.

Elected

1837 MAYO, THOMAS, M.D., F.R.S., the Refuge, Yarmouth, Isle of Wight. S. 1841. C. 1847-8. V.P., 1851-2.

1839 MEADE, RICHARD HENRY, Senior Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1865 MEDWIN, AARON GEORGE, M.D., 11, Montpellier row, Blackheath, Kent.

1867 MEREDYTH, COLOMIATI, M.D., 76, Margaret street, Cavendish square.

1837 MERRIMAN, SAMUEL WILLIAM JOHN, M.D., Consulting Physician-Accoucheur to the Westminster General Dispensary; Marlboro' House, Sandown, Isle of Wight. C. 1851-2. *Trans.* 1.

1852 MERRYWEATHER, JAMES, Consulting Surgeon to the National Dental Hospital; 25, Brook street, Grosvenor square.

1847 MERYON, EDWARD, M.D., F.G.S., *Vice-President*; 14, Clarges street, Piccadilly. L. 1859-60. C. 1864-5. V.P. 1868. *Trans.* 2.

1815 MEYER, AUGUSTUS, M.D., St. Petersburgh.

1868 MICHELL, WILLIAM DANIEL, 38, Delancey street, Gloucester Gate, Regent's park.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Infirmary; Temple row, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD, late of Richmond, Surrey.

1860 *MILES, HERBERT CHALMERS, Assistant-Surgeon in the Royal Horse Artillery, India (Naval and Military Club, Cambridge House, Piccadilly).

1818 *MILLER, PATRICK, M.D., F.R.S.E., Senior Physician to the Devon and Exeter Hospital, and to St. Thomas's Hospital for Lunatics; the Grove, Exeter, Devonshire.

1863 MONRO, HENRY, M.D., Physician to St. Luke's Hospital; 13, Cavendish square. C. 1868.

1844 MONTEFIORE, NATHANIEL, 36, Hyde park gardens.

1848 MOORE, CHARLES HEWITT, *Treasurer*, Surgeon to the Middlesex and St. Luke's Hospitals; 102, Piccadilly. L. 1858. S. 1859-62. C. 1864-5. V.P. 1866-7. T. 1868. *Trans.* 10. *Sci. Com.*

1836 MOORE, GEORGE, M.D., Hastings, Sussex.

Elected

1861 MOREHEAD, CHARLES, M.D., Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; 34, Melville street, Edinburgh.

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.B., Physician to the Manchester Royal Infirmary, and Lecturer on Medicine at the Manchester Royal School of Medicine; 3, Gore street, Piccadilly, Manchester.

1851 MOUAT, FREDERIC JOHN, M.D., Surgeon-Major, Bengal Army; late Inspector-General of Gaols in the Lower Provinces of the Bengal Presidency, and Member of the Senate of the University of Calcutta.

1868 MOXON, WALTER, M.D., F.L.S., Assistant-Physician to, and Lecturer on Morbid Anatomy at, Guy's Hospital; 6, Finsbury circus.

1856 MURCHISON, CHARLES, M.D., F.R.S., Senior Physician to the London Fever Hospital; Physician to, and Lecturer on the Practice of Medicine at, the Middlesex Hospital; 79, Wimpole street, Cavendish square. *Trans.* 3.

1847 MURCHISON, SIMON, Bicester, Oxon.

1863 MYERS, ARTHUR B. R., Coldstream Guards' Hospital, Vincent square, Westminster.

1859 NAYLER, GEORGE, Assistant-Surgeon to the Hospital for Diseases of the Skin, Blackfriars; Assistant-Surgeon to the Royal Orthopædic Hospital; 8, George street, Hanover square.

1835 †NELSON, THOMAS ANDREW., M.D., 10, Nottingham terrace, York gate, Regent's park.

1843 NEWTON, EDWARD, 4, Upper Wimpole street. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., Chelmsford, Essex.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants.

1845 NORRIS, HENRY, Charmouth, Dorset.

1847 *NOURSE, WILLIAM EDWARD CHARLES, 11, Marlborough place, Brighton.

1864 NUNN, THOMAS WILLIAM, Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

Elected

1859 *NUNNELEY, THOMAS, Senior Surgeon to the Leeds Eye and Ear Infirmary; Leeds. *Trans.* 3.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1843 O'CONNOR, WILLIAM, M.D., Senior Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.

1858 OGLE, JOHN WILLIAM, M.D., Physician to, and Lecturer on Pathology at, St. George's Hospital; 13, Upper Brook street, Grosvenor square. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; 3, Stewart terrace, Derby.

1860 OGLE, WILLIAM, M.D., *Secretary*, Assistant-Physician to, and Lecturer on Physiology at, St. George's Hospital; 34, Clarges street, Piccadilly. S. 1868.

1850 OLDHAM, HENRY, M.D., Obstetric Physician to, and Clinical Lecturer on Midwifery at, Guy's Hospital; 26, Finsbury square. C. 1865. *Trans.* 1.

1846 *ORMEROD, EDWARD LATHAM, M.D., Physician to the Sussex County Hospital; 14, Old Steine, Brighton. *Trans.* 2.

1847 *PAGE, WILLIAM BOUSFIELD, Surgeon to the Cumberland Infirmary; Carlisle. *Trans.* 2.

1840 †PAGET, JAMES, D.C.L., F.R.S., Sergeant-Surgeon Extraordinary to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; Member of the Senate of the University of London; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. *Trans.* 8. *Sci. Com.*

1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.

1836 PARKER, LANGSTON, Hon. Surgeon to the Queen's Hospital, Birmingham; Colmore row, Birmingham.

1847 PARKER, NICHOLAS, M.D., Paris.

1841 PARKIN, JOHN, M.D., Rome.

1851 PART, JAMES, M.D., 7, Camden road villas (89, Camden road), Camden town.

Elected

1828 †PARTRIDGE, RICHARD, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Surgeon to King's College Hospital, and Professor of Anatomy in King's College, London; 17, New street, Spring gardens. S. 1832-6. C. 1837-8, 1861-2. V.P. 1847-8. P. 1863-4.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Assistant-Physician to, and Lecturer on Physiology and Comparative Anatomy at, Guy's Hospital; 35, Grosvenor street.

1845 PEACOCK, THOMAS BEVILL, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 20, Finsbury circus. S. 1855-6. V.P. 1867. *Trans.* 2.

1864 PEARSON, DAVID RITCHIE, M.D., 23, Upper Phillimore place, Kensington.

1856 PEIRCE, RICHARD KING, 16, Norland place, Notting hill.

1830 PELECHIN, CHARLES P., M.D., St. Petersburg.

1855 *PEMBERTON, OLIVER, Surgeon to the Birmingham General Hospital, and Lecturer on Surgical Pathology at Sydenham College; 18, Temple row, Birmingham. *Trans.* 1.

1844 PETTIGREW, WILLIAM VESALIUS, M.D., Surgeon to the Female Orphan Asylum; 7, Chester street, Grosvenor pl.

1848 PHILLIPS, EDWARD, M.D., F.L.S., Physician to the Coventry and Warwickshire Hospital; Coventry, Warwickshire.

1852 PHILLIPS, RICHARD, 52, Leinster square, Westbourne grove.

1854 PHILLIPS, THOMAS BACON, M.D., Physician to the Brighton and Hove Dispensary; 36, Lansdowne place, Brighton.

1846 PHILP, FRANCIS RICHARD, M.D., Colby House, Kensington, and Sherborne House, Harrogate, Yorkshire.

1867 PICK, THOMAS PICKERING, Demonstrator of Anatomy, and Curator of the Pathological Museum, at St. George's Hospital; 9, Bolton row, May fair.

1851 *PICKFORD, JAMES HOLLINS, M.D., M.R.I.A., 1, Cavendish place, Brighton.

1836 †PIDDUCK, ISAAC, M.D., 22, Montague street, Russell square. *Pro.* 2.

Elected

1841 PITMAN, HENRY ALFRED, M.D., *Treasurer*, Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8.

1850 POLAND, ALFRED, Surgeon to Guy's Hospital, and to the Eye Infirmary attached to the Hospital. C. 1865-6.

1845 POLLOCK, GEORGE DAVID, Surgeon in Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. *Trans.* 2.

1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square.

1843 POPE, CHARLES, M.D., Glastonbury, Somersetshire.

1846 POTTER, JEPHSON, M.D., F.L.S., Physician to the Liverpool General Hospital for Consumption and Diseases of the Chest; 3, Brooklands, Southport, Liverpool.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., Assistant-Physician to the Hospital for Consumption, Brompton; 6, Nottingham place, Marylebone road.

1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; Examiner in Physiology at the University of London; 45, Seymour street, Portman square.

1851 POWER, ROBERT FRANCIS, M.D., 71, Gloucester place, Portman square.

1857 PRIESTLEY, WILLIAM OVEREND, M.D., Physician-Accoucheur to H.R.H. the Princess Louis of Hesse; Professor of Midwifery in King's College, London, and Physician for the Diseases of Women and Children to King's College Hospital; Examiner in Midwifery at the University of London; Consulting Physician-Accoucheur to the St. Marylebone Infirmary; 17, Hertford street, May fair. *Sci. Com.*

1845 PYLE, JOHN, 59, Oxford terrace, Hyde park. C. 1860.

Elected

1850 QUAIN, RICHARD, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. *Trans.* 1. *Sci. Com.*

1835 †QUAIN, RICHARD, F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.

1852 RADCLIFFE, CHARLES BLAND, M.D., Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8.

1857 RANKE, HENRY, M.D., Munich.

1854 RANSOM, WILLIAM HENRY, M.D., Physician to the Nottingham General Hospital; the Pavement, Nottingham.

1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, May fair. *Trans.* 1.

1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.

1857 REES, GEORGE OWEN, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 26, Albemarle street, Piccadilly. *Trans.* 1.

1868 REEVE, JOHN FOSTER, 8, Sackville street, Piccadilly.

1855 REYNOLDS, JOHN RUSSELL, M.D., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; Examiner in Medicine at the University of London; 38, Grosvenor street.

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; 30, Ramsden street, Huddersfield.

1847 RICHARDS, SAMUEL, M.D., 36, Bedford square.

1852 RICHARDSON, CHRISTOPHER THOMAS, M.B., Warcop, Penrith.

1849 *RICHARDSON, WILLIAM, M.D., 25, Gloucester gardens, Bishop's road, Hyde park.

Elected

1845 RIDGE, BENJAMIN, M.D., 21, Bruton street, Berkeley square.

1843 RIDGE, JOSEPH, M.D., 39, Dorset square. C. 1858. *Pro.* 1.

1863 RINGER, SYDNEY, M.D., Professor of Materia Medica in University College, London, and Physician to University College Hospital; Assistant-Physician to the Hospital for Sick Children; 15, Cavendish place, Cavendish square.

1852 ROBERTS, JOHN, M.R.C.P., 56, Grosvenor street.

1855 ROBERTSON, CHARLES ALEXANDER LOCKHART, M.D., Medical Superintendent of the Sussex County Lunatic Asylum; Hayward's Heath, Sussex.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1862 ROBINSON, CHARLES, F.R.C.P. Edin., 11, Montagu street, Portman square.

1843 ROBINSON, GEORGE, M.D. *Trans.* 2.

1843 RODEN, WILLIAM, M.D., the Grange, Kidderminster, Worcestershire.

1835 †ROE, GEORGE HAMILTON, M.D., Consulting Physician to the Hospital for Consumption and Diseases of the Chest; 124, Park street, Grosvenor square. C. 1841-2. *Trans.* 1.

1836 †ROGERS, ARNOLD, Consulting Surgeon-Dentist to St. Bartholomew's Hospital; 16, Hanover Square.

1829 ROOTS, WILLIAM SUDLOW, F.L.S., Surgeon to the Royal Establishment at Hampton Court; Kingston, Surrey.

1850 ROPER, GEORGE. (China.)

1855 ROSCOW, THOMAS TATTERSALL, M.D.

1836 *ROSE, CALEB BURRELL, F.G.S., 25, King street, Great Yarmouth, Norfolk. *Trans.* 1.

1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary; High street, Hampstead. *Trans.* 1.

1849 ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Trans.* 1.

1863 ROWE, THOMAS SMITH, M.D. Surgeon to the Royal Seabathing Infirmary; Cecil street, Margate, Kent.

1834 RUMSEY, HENRY WYLDBORE, Wolseley House, Cheltenham.

Elected

1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital, and Lecturer on Pathology and Therapeutics at Sydenham College; 91, New Hall street, Birmingham.

1868 RUSSELL, WILLIAM ALEXANDER, L.R.C.P. Ed.; St. Albans, Herts.

1851 SALTER, HENRY HYDE, M.D., F.R.S., Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; 14, Harley street, Cavendish square. S. 1866. C. 1868. *Sci. Com.*

1856 SALTER, SAMUEL JAMES A., F.R.S., F.L.S., Surgeon-Dentist to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. *Trans.* 2.

1849 SANDERSON, HUGH JAMES, M.D., Physician to the Hospital for Women; 26, Upper Berkeley street, Portman square.

1855 SANDERSON, JOHN BURDON, M.D., F.R.S., Assistant-Physician to, and Lecturer on Physiology at, the Middlesex Hospital; Physician to the Hospital for Consumption, Brompton; 49, Queen Anne street, Cavendish square. *Trans.* 1. *Sci. Com.*

1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.

1847 SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Lecturer on Mental Diseases at University College, London; Sandywell park, near Cheltenham.

1845 SAUNDERS, EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; 66, Brook street, Grosvenor square. *Trans.* 3. *Sci. Com.* 2.

1853 SCHULHOF, MAURICE, M.D., Physician to the Royal General Dispensary, Bartholomew Close; 46, Brook street, Grosvenor square.

1861 *SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary; 12, New North Road, Huddersfield.

Elected

1858 SCRATCHLEY, GEORGE, M.D., New Orleans, Louisiana, U.S. [per Arthur Scratchley, Esq., 8A, Waterloo place, Pall Mall].

1863 SEDGWICK, WILLIAM, Surgeon to the St. Marylebone Provident Dispensary; 12, Park place, Upper Baker street. *Trans.* 1.

1856 SERCOMBE, EDWIN, Surgeon-Dentist to St. Mary's Hospital; 41, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1837 †SHARPEY, WILLIAM, M.D., F.R.S., LL.D., Professor of Anatomy and Physiology in University College, London; Member of the Senate of the University of London; Secretary of the Royal Society; University College, and Lawnbank, Hampstead. C. 1848-9. V.P. 1862.

1836 †SHAW, ALEXANDER, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 40, Abbey road west, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Trans.* 4.

1848 *SHEARMAN, EDWARD JAMES, M.D., Moorgate, Rotherham, Yorkshire.

1859 SIBLEY, SEPTIMUS WILLIAM, Lecturer on Pathological Anatomy at the Middlesex Hospital; 12, New Burlington street. *Trans.* 4. *Sci. Com.*

1849 SIBSON, FRANCIS, M.D., F.R.S., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Member of the Senate of the University of London; 59, Brook street, Grosvenor sq. C. 1863-4. *Trans.* 1. *Sci. Com.*

1848 SIEVEKING, EDWARD HENRY, M.D., Physician in Ordinary to H.R.H. the Prince of Wales; Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. *Trans.* 2. *Sci. Com.*

1842 SIMON, JOHN, F.R.S., D.C.L., Surgeon to, and Lecturer on General Pathology at, St. Thomas's Hospital; Medical Officer of the Privy Council; 8, Richmond Terrace, Whitehall, and 40, Kensington square. C. 1854-5. V.P. 1865. *Trans.* 1.

Elected

1865 SIMS, J. MARION, M.D., Paris.

1857 SIORDET, JAMES LEWIS, M.B., Mentone.

1824 †SKEY, FREDERIC CARPENTER, F.R.S., Consulting Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 24, Mount street, Grosvenor square. C. 1828. L. 1829-31. V.P. 1841-2. P. 1859-60. *Trans.* 1.

1852 SMITH, CHARLES CASE, Consulting Surgeon to the Suffolk General Hospital.

1866 SMITH, HEYWOOD, M.A., M.B., Oxon., Assistant-Physician to the Hospital for Women; 42, Park street, Grosvenor square.

1835 SMITH, JOHN GREGORY, 13, New Burlington street.

1843 SMITH, ROBERT WILLIAM, M.D., M.R.I.A., Professor of Surgery in the University of Dublin; Surgeon to the Richmond Hospital; 63, Eccles street, Dublin.

1838 SMITH, SPENCER, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; 9, Queen Anne street, Cavendish square. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865.

1863 SMITH, THOMAS, Assistant-Surgeon to St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children; 5, Stratford place, Oxford street. *Trans.* 2. *Sci. Com.*

1864 *SMITH, THOMAS HECKSTALL, Rowlands, St. Mary Cray, Kent.

1845 SMITH, WILLIAM, Sherwood Villa, Pembroke road, Clifton, Bristol. *Trans.* 1.

1847 SMITH, WILLIAM, M.D., Consulting Physician to the Weymouth Infirmary; Weymouth, Dorsetshire.

1850 SMITH, WILLIAM TYLER, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 21, Upper Grosvenor street. C. 1867-8. *Trans.* 2.

1851 SODEN, JOHN, see *Corbet*.

1830 †SOLLY, SAMUEL, F.R.S., *President*, Senior Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Consulting Surgeon to the Royal General Dispensary, Bartholomew Close; 6, Savile row, Burlington gardens. L. 1838-40. C. 1845-6. V.P. 1849-50. P. 1867-8. *Trans.* 6.

Elected

1868 SOLLY, SAMUEL EDWIN, 6, Savile row, Burlington gardens.

1865 SOUTHAM, GEORGE, Surgeon to the Manchester Royal Infirmary; 21, Lever street, and Oakfield, Pendleton, Manchester. *Trans.* 4.

1865 SOUTHEY, REGINALD, M.B., Assistant-Physician, and Medical Registrar, to St. Bartholomew's Hospital; Physician to the Royal General Dispensary; 32, Montague place, Russell square.

1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Albans.

1834 SPARK, JAMES, Italy.

1851 SPITTA, ROBERT JOHN, M.B., Medical Officer to the Clapham General Dispensary; Clapham Common, Surrey. *Trans.* 1.

1843 *SPRANGER, STEPHEN, Hursley, Hampshire.

1867 SQUAREY, CHARLES EDWARD, Resident Medical Officer, University College Hospital. *Trans.* 2.

1851 STARTIN, JAMES, Surgeon to, and Lecturer on Cutaneous Disorders at, the Hospital for Diseases of the Skin, Blackfriars; 3, Savile row, Burlington gardens.

1854 STEVENS, HENRY, M.D., Lond., Lion Hotel, Shrewsbury.

1842 STEWART, ALEXANDER PATRICK, M.D., *Librarian;* 75, Grosvenor street. C. 1856-7. L. 1863-8.

1859 STEWART, WILLIAM EDWARD, 12, Weymouth street, Portland place.

1856 STOCKER, ALONZO HENRY, M.D., Resident Medical Superintendent of Grove Hall Lunatic Asylum, Bow.

1865 STOKES, WILLIAM, Jun., M.D., 5, Merrion square north, Dublin.

1843 STORKS, ROBERT REEVE, Paris.

1858 †STREATFEILD, JOHN FREMLYN, Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square.

1863 STURGES, OCTAVIUS, M.B., Assistant-Physician to, and Lecturer on Forensic Medicine at, the Westminster Hospital; Physician to the Chelsea, Brompton, and Belgrave Dispensary; 85, Wimpole street, Cavendish square.

Elected

1860 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

1855 SUTTON, JOHN MAULE, M.D.

1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.

1842 SYME, JAMES, D.C.L., F.R.S.E., Surgeon in Ordinary to H.M. the Queen in Scotland; Professor of Clinical Surgery in the University of Edinburgh, and Surgeon to the Edinburgh Royal Infirmary; 1, Shandwick place, Edinburgh. *Trans.* 5.

1854 *SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 32, Beaumont street, Oxford.

1844 TAMPLIN, RICHARD WILLIAM, Surgeon to the Royal Orthopædic Hospital; 33, Old Burlington street.

1848 TANNER, THOMAS HAWKES, M.D., F.L.S., 9, Henrietta street, Cavendish square.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1852 TAYLOR, ROBERT, Surgeon to the Central London Ophthalmic Hospital, and to the Cripples' Home, Hill street; 7, Lower Seymour street, Portman square.

1845 TAYLOR, THOMAS, Lecturer on Chemistry at the Middlesex Hospital Medical School; 1, Warwick place, Grove End road, N.W.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's.

1862 THOMPSON, EDMUND SYMES, M.D., Assistant-Physician to the Hospital for Consumption, Brompton; 3, Upper George street, Portman square.

1857 THOMPSON, HENRY, M.D., Physician to, and Lecturer on Materia Medica at, the Middlesex Hospital; 52, Welbeck street, Cavendish square.

1852 THOMPSON, SIR HENRY, Surgeon Extraordinary to H.M. the King of the Belgians; Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; 35, Wimpole street, Cavendish square. *Trans.* 3.

1862 THOMPSON, REGINALD EDWARD, M.D., Medical Registrar to St. George's Hospital; Physician to the St. George's and St. James's Dispensary; 21, South street, Park lane. *Trans.* 1. *Sci. Com.*

Elected

1836 THURNAM, JOHN, M.D., Resident Medical Superintendent of the Wilts County Asylum, Devizes, Wiltshire. *Trans.* 4.

1848 TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 60, Grosvenor street.

1867 TONGE, MORRIS, M.D., Assistant-Physician to King's College Hospital; 5, Bolton row, May Fair.

1828 TORRIE, JAMES, M.D.

1867 TROTTER, JOHN WILLIAM, Assistant-Surgeon, Coldstream Guards; Hospital, Vincent square, Westminster.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1864 TUFNELL, THOMAS JOLLIFFE, Examiner in Surgery to the Royal College of Surgeons of Ireland; 58, Lower Mount street, Merrion square, Dublin.

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

1855 TULLOCH, JAMES STEWART, M.D., 1, Pembridge place, Bayswater.

1864 TURNER, GEORGE, 9, Sussex gardens, Hyde park.

1845 TURNER, THOMAS, F.L.S., Consulting Surgeon to the Manchester Royal Infirmary; 77, Mosley street, Manchester.

1806 VAUX, BOWYER, Teignmouth, Devon.

1865 VERNON, BOWATER JOHN, Assistant-Demonstrator of Anatomy, St. Bartholomew's Hospital; 93, Wimpole street, Cavendish square.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Hospital, Lisle street, Leicester square; 141, Regent street.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD.

1841 WADE, ROBERT, Senior Surgeon to the Westminster General Dispensary; 68, Dean street, Soho. *Trans.* 1.

1864 WAITE, CHARLES DERBY, M.B., 3, Old Burlington street.

1868 *WALKER, ROBERT, L.R.C.P. Ed., Surgeon to the Carlisle Dispensary; 25, Lowther street, Carlisle.

1867 *WALLIS, GEORGE, Benet street, Cambridge.

Elected

1861 *Walsh, James, M.D., Staff-Surgeon, R.N., 41, Cathrine street, Limerick, Ireland.

1852 Walshe, Walter Hayle, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 37, Queen Anne street, Cavendish square. *Trans.* 1.

1851 Walton, Henry Haynes, Surgeon to the Central London Ophthalmic Hospital; and Surgeon to St. Mary's Hospital; 1, Brook street, Hanover square. *Trans.* 1. *Pro.* 1.

1852 Wane, Daniel, M.D., 20, Grafton street, Berkeley square.

1821 Ward, William Tilleard.

1858 Wardell, John Richard, M.D., 4, Belmont, Tunbridge Wells.

1846 Ware, James Thomas, Consulting Surgeon to the Finsbury Dispensary, and Hon. Surgeon to the Metropolitan Convalescent Institution; 18, Gordon square.

1818 Ware, John, Clifton Down, near Bristol.

1814 †Ware, Martin, 18, Gordon square. C. 1844-5. T. 1846. V.P. 1853.

1866 Waring, Edward John, M.D. [Agents: Messrs. Grindlay and Co., 55, Parliament street].

1861 Waters, A. T. Houghton, M.D., Physician to the Liverpool Northern Hospital, and Lecturer on Anatomy and Physiology in the Liverpool Royal Infirmary School of Medicine; 27, Hope street, Liverpool. *Trans.* 3.

1837 †Watson, Sir Thomas, Bart., M.D., D.C.L., F.R.S.; Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6.

1861 Watson, William Spencer, M.B., Assistant-Surgeon to the Central London Ophthalmic Hospital; 27, Montague street, Russell square.

1854 Webb, William, M.D., Gilkin View House, Wirksworth, Derbyshire.

1840 Webb, William Woodham, M.D., Cliff House, Kirtley, South Lowestoft, Suffolk.

Elected

1842 WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.

1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square.

1835 †WEBSTER, JOHN, M.D., F.R.S., late Physician to the Scottish Hospital, and Consulting Physician to the St. George's and St. James's Dispensary; 9, Queen street, St. Andrew's. C. 1843-4. V.P. 1855-6. *Trans.* 6. *Pro.* 1.

1844 WEGG, WILLIAM, M.D., Physician to the St. George's and St. James's Dispensary; 26, Park lane. L. 1854-8. C. 1861-2.

1861 WELLS, JOHN SOELBERG, Professor of Ophthalmology in King's College, London, and Ophthalmic Surgeon to King's College Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital; 16, Savile row.

1854 WELLS, THOMAS SPENCER, Surgeon in Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. *Trans.* 6. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Physician to the Hospital for Sick Children; 61, Wimpole street, Cavendish square. C. 1855-6. V.P. 1863. *Trans.* 2. *Sci. Com.*

1828 WHATLEY, JOHN, M.D.

1849 WHITE, JOHN.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1844 WILDBORE, FREDERIC, 1, Trafalgar place east, [245] Hackney road.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., [5, Lincoln's Inn Fields].

1863 WILKS, SAMUEL, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; Examiner in Medicine at the University of London; 11, St. Thomas's Street, Southwark.

1865 WILLETT, ALFRED, Assistant-Surgeon to St. Bartholomew's Hospital; 36, Wimpole Street, Cavendish square.

Elected

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1860 WILLIAMS, ARTHUR WYNN, M.D., Physician to the Samaritan Free Hospital; Physician-Accoucheur to the Western General Dispensary; 1, Montagu square.

1840 WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton; 49, Upper Brook street, Grosvenor square. C. 1849-50. V.P. 1860-1. *Sci. Com.*

1859 *WILLIAMS, CHARLES, Surgeon to the Norwich Dispensary; 9, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.B. Oxon., Assistant-Physician to the Hospital for Consumption, Brompton; 49, Upper Brook street, Grosvenor square.

1859 WILLIAMS, JOSEPH, M.D., 8, Tavistock square.

1868 WILLIAMS, WILLIAM RHYS, M.D., Bethlem Hospital.

1829 WILLIS, ROBERT, M.D., Barnes, Surrey. L. 1838-41.

1839 †WILSON, ERASMUS, F.R.S., Consulting Surgeon to the St. Pancras Infirmary; 17, Henrietta street, Cavendish square. *Trans.* 2.

1863 WILSON, ROBERT JAMES, L.R.C.P. Edin., 24, Grand Parade, St. Leonard's-on-Sea, Sussex.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.

1825 WISE, THOMAS ALEXANDER, M.D., Rostellan Castle, Rostellan, County Cork.

1841 WOOD, GEORGE LEIGHTON, Surgeon to the Bath General Hospital; 27, Queen square, Bath.

1851 WOOD, JOHN, Assistant-Surgeon to King's College Hospital, and Demonstrator of Anatomy in King's College, London; Examiner in Anatomy at the University of London; 4, Montague street, Russell square. C. 1867-8. *Trans.* 2.

1848 WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley Street, Cavendish square. C. 1867-8.

Elected

1833 †WORMALD, THOMAS, Consulting Surgeon to St. Bartholomew's Hospital; 42, Bedford row. C. 1839. V.P. 1854.

1842 WORTHINGTON, WILLIAM COLLINS, Senior Surgeon to the Lowestoft Infirmary: Lowestoft, Suffolk. *Trans.* 3.

1865 WOTTON, HENRY, Jun.

1855 WRIGHT, HENRY G., M.D., Physician to the Samaritan Free Hospital for Women and Children; 66, Harley Street, Cavendish square.

1860 WYATT, JOHN, Surgeon-Major, Coldstream Guards; Hospital, Vincent square, Westminster.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1853 BRODIE, SIR BENJAMIN COLLINS, Bart., M.A., F.R.S., Waynflete Professor of Chemistry at Oxford; Cowley House, Oxford.

1847 CHADWICK, EDWIN, late Commissioner of the Board of Health; Corresponding Member of the Academy of Moral and Political Sciences of the Imperial Institute of France.

1868 DARWIN, CHARLES, M.A., F.R.S., Corresponding Member of the Academies of Sciences of Berlin, Stockholm, Dresden, &c.; Down, Bromley, Kent.

1857 FARR, WILLIAM, M.D., D.C.L., F.R.S., General Register Office, Somerset House, and Southlands, Bromley, Kent.

1841 HERSCHEL, SIR JOHN FREDERICK WILLIAM, Bart., D.C.L., F.R.S., Foreign Associate of the Academy of Sciences of the Imperial Institute of France; Collingwood, near Hawkhurst, Kent.

1868 HOOKER, JOSEPH DALTON, M.D., D.C.L., LL.D., Director of the Royal Botanic Gardens, Kew; Corresponding Member of the Academy of Sciences of the Imperial Institute of France; Kew.

1868 HUXLEY, THOMAS HENRY, LL.D., Professor of Natural History in the Royal School of Mines; Hunterian Professor of Comparative Anatomy in the Royal College of Surgeons; Fullerian Professor of Physiology in the Royal Institution; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, etc.; 26, Abbey place, St. John's Wood.

1868 LYELL, SIR CHARLES, Bart., D.C.L., LL.D., Corresponding Member of the Academies of Sciences of Paris, Berlin, Philadelphia, Boston, &c.; 73, Harley street, Cavendish square.

Elected

1866 MURCHISON, SIR RODERICK IMPEY, Bart., K.C.B., D.C.L., LL.D., M.A., F.R.S., Director-General of the Geological Survey; Foreign Associate of the Academy of Sciences of the Imperial Institute of France; 16, Belgrave Square.

1847 OWEN, RICHARD, D.C.L., LL.D., F.R.S., Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Imperial Institute of France; Sheen Lodge, Mortlake.

1825 SEDGWICK, The Rev. ADAM, A.M., D.C.L., LL.D., F.R.S., Woodwardian Professor of Geology, Cambridge.

1868 TYNDALL, JOHN, LL.D., Professor of Natural Philosophy in the Royal Institution, and in the Royal School of Mines; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Royal Institution, Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1841 ANDRAL, G., M.D., Member of the Institute and of the Imperial Academy of Medicine; Physician in Ordinary to the Emperor of the French; Paris.

1862 CRUVEILHIER, JEAN, M.D., Physician to the "Hôpital de la Charité;" Member of the Imperial Academy of Medicine; Paris.

1864 DONDERS, FRANZ CORNELIUS, M.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1856 DUBOIS, BARON PAUL, Commander of the Legion of Honour; Member of the Imperial Academy of Medicine; late Dean of the Faculty of Medicine; Paris.

1835 EKSTRÖMER, CARL JOHAN, M.D., C.M., K.P.S., and W., Physician to the King of Sweden; President of the College of Health, and Director-General of Hospitals; Stockholm.

1841 EHRENBERG, CHRISTIAN GOTTFRIED, Foreign Associate of the Academy of Sciences of the Imperial Institute of France; Berlin.

1868 GROSS, SAMUEL D., M.D., F.R.C.P. Philad., Professor of Surgery in the Jefferson Medical College of Philadelphia.

1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.

1859 HENLE, J., M.D., Professor of Anatomy at Göttingen.

1868 KÖLLIKER, ALBERT, Professor of Anatomy at Würzburg.

1856 LANGENBECK, BERNHARD, M.D., Professor of Surgery in the University of Berlin.

1868 LARREY, HIPPOLYTE BARON, Member of the Institute; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Surgeon-in-Ordinary to the Emperor; Commander of the Legion of Honour, &c.; Rue de Lille 91, Paris.

Elected

1843 LIEBIG, BARON JUSTUS von, M.D., Conservator of the Royal Collection, and Professor of Chemistry, in the University of Munich; Foreign Associate of the Academy of Sciences of the Imperial Institute of France; Munich.

1841 LOUIS, P. C. A., M.D., Honorary Physician to the Hotel-Dieu; Member of the Imperial Academy of Medicine; Paris.

1868 NÉLATON, AUGUSTE, Member of the Institute, and of the Imperial Academy of Medicine; Surgeon to the Emperor; Member of the Senate; late Professor of Clinical Surgery to the Faculty of Medicine; 1, Avenue d'Antin, Paris.

1862 PIROGOFF, NIKOLAUS, M.D., Professor of Surgery to the Medico-Chirurgical Academy in St. Petersburg, and Director of the Anatomical Institute; Consulting Physician to the Hospitals Obuchow, Peter-Paul, and Maria Magdalena; St. Petersburg.

1850 ROKITANSKY, CARL, M.D., Curator of the Imperial Pathological Museum, and Professor of the University of Vienna. Referee for Medical and University Education to the Austrian Ministry; Vienna.

1856 STROMEYER, LOUIS, M.D., Director-General of the Medical Department of the Army of Hanover; Hanover.

1856 VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Imperial Institute of France; Berlin.

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ON SOME

ANALOGIES OF CHOLERA,

IN WHICH

SUPPRESSION OF URINE IS NOT ACCOMPANIED BY SYMPTOMS OF URÆMIC POISONING.

BY

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THE indirect aid afforded by analogy in the investigation of disease may be said to correspond with that of a reflecting mirror, in which there is presented a more or less clearly defined image of what would otherwise be obscure or hid; and, therefore, as an assistance towards lessening the difficulty which attends the investigation of cholera, which, although uniformly well marked as regards its symptoms, is exceptionally obscure as regards its nature, analogy, in so far as it enables us to trace out and establish a resemblance between it and other morbid conditions of the system, which admit of being more readily explained, will be useful, provided the principle be sound, on which the analogical reasoning is based. For it has become a necessary and, in some respects, an unpleasant duty to direct attention to the fact that, in the investigation of this as of other baffling diseases, attempts have on many occasions been made to establish false analogies in support of unfounded theories of the disease, or of ill-

devised plans of treatment, which, after attracting popular attention, have for the most part been soon slighted and forgotten. It may, moreover, be remarked that in some cases the attempt to establish an analogy, even if successful, would not be attended with any useful result, for with respect to our knowledge of the nature of the disease, it would be but the substitution of one difficulty for another; as, for example, when cholera is compared with tetanus, or alleged to be an epilepsy of the organic nervous system, or the cold stage of an incomplete attack of ague. In like manner the analogies which have been urged in support of some of the proposed remedies for the disease are equally defective in the information they are intended to convey, as when cholera is compared with sea-sickness, in consequence of its having been assumed that both admit of being prevented and cured by ice to the spine; or when, in support of the castor-oil or eliminative method of treatment, cholera is compared with smallpox. Even the term eliminative, as used in this latter case, is objectionable, since there is nothing in common between the two processes by which the characteristic discharge in smallpox is excluded from the system by the skin, or the characteristic discharge in cholera by the gastro-intestinal mucous membrane; and if, in support of this theory of so-called elimination, and apart from any preconceived theory of treatment, it were desirable, in consequence of the sympathetic connection which exists between the skin and the alimentary mucous membrane, to establish an analogy between cholera and some disease characterised by abnormal and excessive discharge from the skin, the most appropriate for that purpose would decidedly be the sudor anglicus, or sweating sickness of the Middle Ages, which has apparently prevailed though to a less noticeable extent in modern times, and which Dr. Roupell[1] has referred to as approaching nearer than any other epidemic disease to cholera. But it must be confessed that inquiry even in this direction, notwithstanding the parallelism presented by the profuse and characteristic discharges, could not be expected to lead to

[1] 'On Cholera' (the Croonian Lectures for 1833), p. 72.

any useful result, in consequence of the sudor anglicus being quite as great if not a greater pathological mystery than cholera itself. In order, therefore, to avoid the errors above referred to, and to check the ever-recurring tendency, in a discussion of this kind, to introduce assumption in the place of fact, it is proposed in the following attempts to trace an analogy between cholera and allied but less obscure affections of the system, to limit the inquiry to a comparison between the well-defined and characteristic group of symptoms and pathological changes which accompany the collapse of this disease, with special reference to suppression of urine during life, and an empty and contracted state of the bladder after death, and such cases of collapse resulting either from poison or from disease, in which a corresponding condition has been developed and observed.

It is a noteworthy fact that when cholera first attracted general attention, and before the public mind had become familiarised with the peculiar and fatal character of the disease, it was very commonly ascribed to irritant poisoning, and popular tumults were in many places the result of this belief. It is of some importance with reference to the present inquiry to remark that this now almost forgotten but once widespread delusion was suggested by the analogy which prevails between the chief symptoms of cholera and many of those poisons, the effects of which are more or less commonly known; and that, like other errors of a similar kind, the hasty and imperfect reasoning on which it was based contained, as it were, the germs of truth. For if we proceed to analyse the earliest of the analogies of cholera, it will be found that most of the symptoms which are met with in the collapsed stage of the disease occur also in the case of certain poisons, the action of which is essentially referable to the gastro-intestinal mucous membrane; and it may in consequence be not unreasonable to expect that if the inquiry be pursued in this direction, it will furnish us with a clue to the primary seat of the disease. In addition, however, to exciting early and popular attention, this analogy between cholera and cases of irritant poisoning seems to

have specially attracted the notice of many scientific observers in subsequent epidemics of the disease, by whom the importance of urging the inquiry in this direction has been fully recognised. In an able article on "Diseases which may simulate Poisoning," Devergie[1] assigns the first place to cholera, and mentions suppression of urine as one of the symptoms common to the collapse of both. Tardieu[2] has also published numerous illustrations in support of this statement. And among other observers whose evidence might be cited on this occasion is Dr. Horace Jeaffreson, who has had favorable opportunities for studying cholera at the London Hospital during the outbreak of the disease in the eastern districts of London in 1866; and who, in a lately published and instructive paper "On the Pathology of Cholera Collapse"[3] has justly remarked, that "inquiry in this direction will give us some remarkable analogies."

In this preliminary stage of the investigation it will be sufficient to state that the cases in which such an analogy can be more or less clearly established, are acute poisoning by corrosive sublimate, by arsenic, and by mineral acids, especially nitric acid; the effects which follow the eating or drinking of poisonous animal matters, such as tainted or simply unwholesome meat or fish, and milk which has undergone some injurious but as yet unknown change, decomposing vegetables and some of the poisonous fungi; and the excessive action of certain drugs, for the most part belonging to the class of drastic purgatives, especially croton oil. After a brief consideration of some of the more important cases of this description, in which the collapse resulting from the gastro-intestinal action of an irritant poison has been noted as closely analogous to that of cholera, attention will be directed to a well-defined group of cases in which the symptoms from disease referrible to the same part of the system so far simulate choleraic collapse, as to have been occasionally and almost unavoidably mistaken for it.

[1] 'Médecine Légale,' 3e édit., tom. iii, chap. xxi, 1852.

[2] 'Etude Médico-Légale et Clinique sur l'Empoisonnement,' 1867.

[3] 'Edinburgh Medical Journal,' Dec., 1866, pp. 520-538.

In cases of poisoning by corrosive sublimate, both the symptoms during life and the appearances after death are in many important particulars, and especially as regards the urinary secretion, closely analogous to those of cholera. This is well shown in the report of a case recorded by the late Dr. William Henry,[1] of Manchester, in which a woman, aged twenty years, died from the effects of this poison on the sixth day, in which it is mentioned that "no urine was voided after the third day; and on introducing the catheter repeatedly, the bladder was found empty;" and that after death "the bladder was empty and exceedingly contracted." In five cases in one family, recorded by the late Mr. J. W. Valentine,[2] of Bolsover, in which a mother and three children died, and one child recovered from poisoning by corrosive sublimate, suppression of urine was in like manner a characteristic symptom in the collapse, which in other respects also closely resembled that of cholera. In two of the fatal cases, the urinary bladder was contracted to "the size of a walnut;" in a third case, it was of very small size, and empty; and in a fourth case it was found "closely contracted to the size of a marble." The child who recovered was a girl, aged fourteen years, who swallowed only a small portion of the poison, and had suppression of urine for three days. Dr. Robert Venables[3] has recorded a case of poisoning by corrosive sublimate, in which a servant girl died from the effects of the poison on the eighth day. In this case it was specially noted that "there was a total and permanent suppression of urine; and that there might be no room for doubt upon this subject afterwards, Mr. Marten, at my suggestion (adds Dr. Venables), introduced the catheter, but no urine flowed." After death "the bladder was (found) perfectly empty, and very much contracted in size." In a case of poisoning with corrosive sublimate, reported by Mr. Archibald Blacklock,[4] of Dumfries, in which a man, aged

[1] 'Edinburgh Medical and Surgical Journal,' vol. vii, 2nd edit., pp. 150, 151, 1811. [2] Ibid., vol. xiv, pp. 468-474, 1818.

[3] 'London Medical Gazette,' vol. viii, pp. 616-623, 1831.

[4] 'Edinburgh Medical and Surgical Journal,' vol. xxxvi, pp. 92-4, 1831.

fifty years, died on the fifth day; the urine was suppressed from the time the poison was taken, and we are informed that there was no inclination to pass it even when at stool. The pulse in this, as in many other cases recorded, was almost imperceptible for some hours before death. In Mr. (now Sir James) Syme's 'Clinical Report for the Winter Session 1834-5,'[1] there is the case of a man named James Maxwell, aged thirty-five years, who suffered severely, but did not die from the poisonous effects of mercury externally applied. In this case the urine was suppressed for five days, in consequence of which "the catheter was introduced repeatedly without allowing any to escape, except two or three teaspoonfuls of mucous-looking fluid destitute of urinous smell." * * * "The patient drank freely during the suppression of urine; he was quite sensible; and lay quiet without any disposition to coma." In commenting on this case, Mr. Syme remarks that it "is interesting on several accounts. In the *first* place, it affords an example of the suppression of urine, which results from poisonous doses of the oxymuriate of mercury given internally, occurring from the external application of a mercurial salt; *secondly*, the suppression of urine was not accompanied with coma; and *thirdly*, the patient recovered after suffering complete suppression of urine for five days." In a case of acute poisoning by corrosive sublimate, which was under observation in Guy's Hospital in February, 1843, it is recorded that there was "complete suppression of urine, and symptoms closely resembling those of cholera." * * * The patient lived four days, and did not pass any urine during the whole of this time;" and after death "the bladder was empty and contracted."[2] According, also, to Dr. Copland,[3] it appears that in this form of poisoning there is suppression of urine or dysuria during life, and that after death "contraction of the urinary bladder is always observed." Ample evidence has, moreover, been cited by Dr. Taylor[4] to show that sup-

[1] 'Edinburgh Medical and Surgical Journal,' vol. xliv, pp. 26, 27, 1835.

[2] 'Guy's Hospital Reports,' vol. ii, pp. 24-27, 1844.

[3] 'Dictionary of Practical Medicine,' vol. iii, part i, pp. 349, 350.

[4] 'On Poisons,' 2nd edit., pp. 446-459, 1859.

pression of urine, which, when unaccompanied by head-symptoms, has been thought to be almost peculiar to choleraic collapse, is a frequent result of poisoning by corrosive sublimate, both in those cases in which the poison has been swallowed, and in those in which it has been externally applied.

Among continental observers whose authority might be cited on this subject, is Casper,[1] who has specially noted that in cases of poisoning by corrosive sublimate, there is "suppression of urine;" and after death he has also found "the urinary bladder small and contracted." In like manner, Flandin[2] includes among the signs of poisoning by mercury "scantiness or total suppression of urine;" and in his summary of the comparative effects of mercury and arsenic (in poisonous doses) on the animal economy, which present, as he justly remarks, "great analogies," adds that "arsenic, in paralysing nervous action, diminishes or suspends the secretion and the emission of urine. Mercury produces the same effect; but, moreover, it irritates the bladder and urethra, and gives rise to fictitious and painful efforts to pass water."[3] In one of the cases recorded by this observer,[4] in which a young girl died from poisoning by corrosive sublimate on the third day, there was complete and prolonged suppression of urine, which was demonstrated by passing a catheter on the first, and again at the end of the second day, on both of which occasions no urine flowed. In another case recorded by the same observer,[5] in which a lad, aged fifteen years, died five days and six hours after poisoning by corrosive sublimate, it was ascertained that "the urinary secretion, during all this time, was suspended," and the bladder after death was found to be contracted. Lastly, the evidence which has been published on this subject by Tardieu[6]

[1] 'A Handbook of the Practice of Forensic Medicine,' Sydenham Society, vol. ii, p. 62, 1862.

[2] 'Traité des Poisons,' tom. ii, p. 140, 1853.

[3] Ibid., p. 113.

[4] Ibid., pp. 114-116.

[5] Ibid., pp. 116, 117.

[6] Op. cit., 1867. See Obs. 2, p. 596; Obs. 4, p. 597; Obs. 5, p. 598; and Obs. 8, p. 605.

may be referred to as very conclusive in favour of the collapse from poisoning by corrosive sublimate being associated with suppression of urine, and other symptoms similar to those of cholera.

It is important, moreover, to notice that although in the preceding cases there was suppression of urine during collapse from this poison in the human subject, yet the opposite condition of a copious and free discharge of urine has been observed to result from corrosive sublimate given in large quantities to horses. This apparent discrepancy in the effect of the poison on the urinary secretion in men[1] and horses is not due, as perhaps might be hastily assumed, to difference in the composition of their urine, but chiefly, if not altogether, to the fact (which it would be useful to consider in connection with the comparative influence of epidemic cholera on man and the lower animals) that corrosive sublimate has no irritant effect on the stomach of the horse corresponding with what has been observed in man; and that it does not produce any analogous collapse. This has been satisfactorily established by the experiments of the late Mr. Stevenson,[2] veterinary surgeon, of Norwich, who found that considerable quantities of corrosive sublimate might be given with impunity to a horse; and that in this animal neither disorganization nor inflammation of the coats of the stomach

[1] In connection with this subject it should be mentioned that in a case observed by Mr. John Ward, of the Royal Cornwall Militia, in which two brothers were poisoned by the external application of corrosive sublimate, in one of them, aged 24 years, death occurred on the eleventh day, and it is stated that he passed urine freely on different occasions during the illness; but there was no opportunity of knowing what was the condition of the gastro-intestinal mucous membrane, as no examination of his body after death was made. In the other brother, aged 19 years, death occurred on the fifth day; the symptoms of collapse were more acute than in the former case, and throughout the illness the urine was completely suppressed, none being obtained on passing a catheter. After death the stomach was found highly inflamed and ulcerated, and the bladder was "healthy in appearance, but did not contain the least drop of urine."—('London Medical Gazette,' vol. iii, pp. 666-667, 1829.)

[2] 'Edinburgh Medical and Surgical Journal,' vol. v, pp. 254-256, 1809.

resulted from repeated doses, varying from two drachms to two ounces, of the poison.

In cases of poisoning by arsenic, as in the preceding class of cases, it has been observed that, in addition to the more common results of poisoning, there are others which sometimes closely assimilate its effects to cholera; in consequence of which Devergie[1] refers to it as "one of the poisons which best simulates cholera;" whilst Tardieu[2] emphatically states that of the spontaneous diseases most closely allied with arsenical poisoning, "the first of all is incontestably cholera." Among the symptoms and pathological appearances which have been noticed in some of these cases, and to which more especially attention should be directed, are suppression of urine, cyanosis, conjunctival injection, and a physical alteration in the condition of the blood closely analogous to that which occurs in cholera; together with such a failure of the circulation that, occasionally even for some hours before death, neither pulse nor beating of the heart can be perceived: in addition to which Tardieu[3] mentions that there is sometimes "a serous and white diarrhœa." In a case of this description, reported by Dr. Wilks,[4] the patient was a man, æt. 50, who was admitted into Guy's Hospital on May 3rd, 1855, and who died three hours and a half after admission, and nine hours after taking the poison. The symptoms throughout closely resembled those of cholera; the urine was suppressed, and after death the bladder was observed to be firmly contracted, and it contained only two drachms of opaque urine. Dr. Wilks, in commenting on this case, remarks, "it would have required more than ordinary observation and skill at such a time, and in the absence of a history, to have avoided a suspicion that the patient was not the subject of this disease (cholera). The collapse, the pulselessness, the coldness of the surface, the lividity, the restlessness, the violent cramps, and, moreover, the character of the stools (which might have passed well for choleraic after the ad-

[1] Op. cit., p. 774.
[2] Op. cit., p. 337.
[3] Op. cit., p. 328.
[4] 'Guy's Hospital Reports,' 3rd ser., vol. i, p. 364, 1855.

ministration of the eggs) all closely resembled the symptoms of that malady. The post-mortem examination also further carried out the similarity, and probably would have confirmed the diagnosis of cholera, if it had been superficially made, and the stomach had not been examined as is sometimes the case." Dr. Wilks in a note informs us that "during the epidemic of 1849, a practitioner in the neighbourhood of the hospital mistook, during the life of the patient, a case of arsenical poisoning for one of cholera, and his faulty diagnosis was severely animadverted upon by the coroner's jury." Tardieu,[1] in a paper on cases of death from natural causes which may be attributed to poisoning, mentions a mistake of an opposite character to that referred to by Dr. Wilks, in which a case of cholera, occurring in 1849, was at first assumed to be a case of suicide by arsenical (Schweinfurt's) green. A well-marked suicidal case of arsenical poisoning simulating cholera has been recorded by M. Flandin,[2] in which a prisoner on hearing his condemnation to death, swallowed twelve grammes of arsenious acid, and died in thirteen hours. There was immediate and violent vomiting and pain; and within little more than two hours after swallowing the poison the pulse was imperceptible, not the least movement of the artery being detected, and not the slightest beating of the heart could be felt on applying the hand to the præcordial region. The surface of the body became icy and cyanosed; whilst the intellectual faculties, up to the last hour, remained unimpaired. With regard to the urinary secretion it was noted that the patient, in the course of the illness, experienced a desire to pass water, but without the ability to satisfy it; and when the catheter was passed only a few spoonfuls of *clear urine* were obtained. After death the stomach was found to be in a state of complete disorganization; the bladder contained about a "glass of urine," which presented no special character; and it was moreover remarked that all the divisions of the pulmonary artery were filled with incoagulable blood, whilst, on the contrary, the

[1] 'Annales d'Hygiène Publique,' 2e sér., tom. ii, pp. 150-178, 1854.

[2] Op. cit., tom. i, pp. 494-501.

pulmonary veins were nearly empty. The same observer has recorded a case[1] of arsenical poisoning through a wound in the skin, in which the patient died seven days after the application of the poison. The intelligence in this case was preserved up to the last moment of life; the conjunctivæ were injected with blood; and the urinary secretion was very scanty. After death the bladder was found to be contracted and empty; and "the lungs, collapsed above and behind, presented hypostatic sanguine congestion at their base," similar to what is usually observed after death from choleraic collapse. In another case of "acute poisoning by cutaneous absorption" of arsenic, recorded by Flandin,[2] it was noted on the day following the poisoning that the urine, "since the beginning of the illness, was totally suppressed;" and after death, which occurred on the sixth day, the bladder was found to be empty; it is not, however, distinctly stated that the suppression of urine continued during the whole of the six days. The chief effects of the poison absorbed in this case appear to have been on the alimentary canal at the junction of the duodenum with the jejunum, where the ulceration had led to perforation. Among other cases of poisoning by arsenic to which reference might with advantage be made, are Casper's case No. 185,[3] in which death occurred in twenty-four hours, and the urinary bladder was found to be empty; M. Delsol's[4] case, in which there was marked cyanosis as well as suppression of urine; and some cases recorded by Tardieu,[5] including that of the duc de Praslin.

With reference to the suppression of urine, which has been noted in the preceding and in other cases of arsenical poisoning, it is necessary to state that very opposite opinions have been published on the subject; for whilst Devergie[6] mentions "complete suppression of the emission of urine," as one of

[1] Op. cit., tom. i, pp. 545-551.

[2] Ibid., pp. 502-509.

[3] Op. cit., pp. 72, 73.

[4] 'Bull. de la Soc. Anatom. de Paris,' 2e série, tom. vii, pp. 362-364, 1862.

[5] Op. cit., 1867, pp. 379-380, 384-385, and 385-401.

[6] Op. cit., tom. iii, pp. 505 and 552-553.

the symptoms produced by this poison; and especially refers to the re-establishment of the urinary secretion as the turning point in favour of recovery in the case of a woman whose appearance had previously been such that "one would have said she had been attacked with most violent cholera;" and, in like manner, Flandin,[1] supported by the evidence of many other observers, includes suppression of urine "among the general physiological effects" of poisoning by arsenic, and further remarks that it has been noted at all times and by all observers: on the other side, Orfila stated at the celebrated trial of Madame Leffarge that the flow of urine in these cases is absolutely free;[2] and Dr. Taylor[3] has remarked that, "with respect to the urinary secretion (in cases of poisoning by arsenic) there is no certain rule; it is sometimes suppressed, as in several cases reported by Flandin; at other times it is natural or only slightly diminished." This apparently conflicting testimony admits of being to some extent if not altogether accounted for by the fact that arsenic has a two-fold action on the living body, and produces, as Dr. Christison[4] assures us is now generally acknowledged, two classes of phenomena. One action, which is local and purely irritant, affects chiefly that portion of the alimentary mucous membrane which corresponds with what appears to be the primary seat of disease in cholera; and this action is manifested both in those cases in which the poison is swallowed and consequently applied directly to the mucous membrane, and in those in which it enters the system through the skin.

[1] Op. cit., tom. i, p. 514.

[2] In the latest publication of Orfila "On Poisoning by Arsenic" ('Dictionnaire Encyclopédique des Sciences Medicales,' tom. vi, p. 224, 1867), he informs us that there have been more frequently than otherwise noted "disorders which are observed in the algide period of cholera, smallness and irregularity of the pulse, coldness of the skin, prostration, cramps, cyanosis;" and, with regard to the urinary secretion, that "the emission of urine is in general suspended, but the secretion is only diminished, and, save in exceptional circumstances, it is possible to excite the urinary secretion by means of diuretic drinks."

[3] 'On Poisons,' 2nd edit., p. 359, 1859.

[4] 'A Treatise on Poisons,' 4th edit., p. 288, 1845.

The other action is of a more complex character, producing results which are to some extent secondary and remote; for whilst a state of collapse resembling that of cholera is caused by the irritant effects of the poison on the abdominal nervous system, injurious effects are produced on organs remote from the primary seat of mischief, through the irritant effects of the poison on the circulating blood. In the one case the irritant action of arsenic is circumscribed, and, in affecting chiefly that portion of the alimentary mucous membrane which corresponds with what may with great probability be assumed to be the primary seat of disease in cholera, produces, through the influence of the abdominal nervous system, a condition analogous to choleraic collapse; in the other case, through its presence in the blood, secondary and remote effects are produced beyond those which are developed in that disease.

The recognised difference in the action of arsenic on the animal economy may perhaps to some extent account for the contradictory character of some of the experimental evidence derived from the effects of arsenic on horses and dogs; although part of the difference which has been observed in these cases may possibly be due to arsenic, like corrosive sublimate, not having the same poisonous influence on man and the lower animals. For M. Delafont[1] in his elaborate memoir, in answer to the question "Is the urinary secretion suppressed in acute poisoning by arsenious acid?" which was read at the French Academy of Medicine, on July 5th, 1842, states, as the results of twelve experiments with arsenic on horses and dogs, that "in none of the animals submitted to the experiment has the secretion of urine been suppressed." At the same time it was observed that the urine passed in all these cases of fatal poisoning contained arsenic.

Besides the influence of this poison on the secretion of urine, the effects of arsenic on the vascular system and the blood afford further corroborative evidence in favour of the

[1] 'Gazette Médicale de Paris,' 2e sér., tom. x, p. 446, 1842.

analogy with cholera. Casper[1] has remarked that "the blood generally, in acute arsenical poisoning, is deficient in coagulating power, and never forms a firm clot;" which accords with the experiments of Brodie,[2] and the evidence[3] also of Morgagni, Ruysch, and other observers. Novati,[4] who has had the opportunity of opening the bodies of many individuals poisoned by arsenic, and has carefully studied the physical and physiological characters of this form of poisoning, states "that the blood in these patients is liquid and black as it is in cholera," and without any trace of coagulation. "It should not (Novati very justly adds) be forgotten meanwhile, 1st, that many diseases may give to the blood characters analogous to this; 2nd, that other poisonous substances will be capable of giving the same result; and 3rd, that when death has not occurred promptly, the inflammatory reaction determined by the eschars may quite change these characters of the blood." Lastly, both Orfila and Flandin have cited, with reference to the same subject, the evidence of Rognetta and other members of the Italian school, who agree in stating that "the blood in poisoning by arsenic is deliquescent, syrupy, and nearly the same as in cholera; and this state is not the result of a direct chemical action on the blood; it is altogether a physiological effect, a consequence of the hyposthenic action of the arsenic;" such hyposthenic action being referrible to the lowering influence of the poison on the nerves supplied to the vascular system, and not to any specific effect of arsenic on the blood. And this theory of action quite agrees with the latest opinion of Orfila[5] on the subject, who states that "at the present day, after the beautiful researches of M. Claude Bernard and the ingenious experiments of M. Marey, establishing that inflammation and fever are associated with a state of depression or

[1] 'Op. cit., vol. ii, p. 55.

[2] 'Phil. Trans.,' 1812, part i, p. 214.

[3] Cited by Orfila, 'Traité de Toxicologie,' 4e édit., tom. i, p. 344, 1843.

[4] Cited in 'Gazette Médicale de Paris,' 3e sér., tom. vi, p. 571, 1838.

[5] 'Dictionnaire Encyclopédique des Sciences Médicales, tom. vi, p. 224, 1867.

paralysis of certain parts of the great sympathetic, one would not know how to consider the action exercised by arsenious acid otherwise than as an action really hyposthenic."

With respect to poisoning by the mineral acids, it has been specially noted in some of the more carefully recorded cases that, apart from any absorption of the acid and consequently abnormal irritation of secreting organs from its presence in the blood, the corrosive action of the acid on the gastro-intestinal mucous membrane and the resulting collapse lead to the urine being suppressed: and this analogical evidence is the more valuable from the fact that the suppression in cases of this description cannot be accounted for by a complementary discharge from the bowels, as there is usually obstinate constipation at the same time.

In a case of poisoning by hydrochloric acid, reported by Dr. Collas,[1] in which the patient was a Hindoo, æt. 28, in addition to coldness of the skin, small and frequent pulse, and other symptoms of collapse, "there was suppression of urine, but no purging." And Devergie[2] cites the case of a boy, æt. 3½, who was poisoned by hydrochloric acid, and in which after death it was noted that "the bladder contained no urine."

Among the symptoms of poisoning by sulphuric acid, Tardieu[3] mentions that "the urine is suppressed," and that after death "the bladder is usually empty." Casper[4] has recorded the case of a boy, æt. 16 (No. 194), in which the stomach internally appeared quite black, and perforation had occurred. There is no allusion in this case to the urinary secretion during life, but it was observed after death that the urinary bladder was empty; that the lungs were remarkably anæmic; and that the whole of the blood in the body, as is usual in such cases, was dark and syrupy. In Casper's case (No. 197) of a girl, æt. 20, the lungs were anæmic, and the bladder

[1] 'Annales d'Hygiène,' Janvier, 1858, p. 209, cited by Dr. Taylor, 'On Poisons,' 2nd edit., p. 290, 1859.

[2] Op. cit., tom. iii, pp. 290-296, 1852.

[3] Op. cit., pp. 175 and 180.

[4] Op. cit., vol. ii, 1862.

empty; and again in a case (No. 198) recorded by the same observer, of a child, aged six weeks, it is stated that the urinary bladder was empty and that the lungs contained no blood. Mr. Stanski[1] and Mr. Robinson[2] have also recorded cases in which the symptoms of poisoning by this acid were analogous to those of cholera.

From the elaborate researches of M. Tartra[3] on poisoning by nitric acid, it appears that the corrosive action of this poison is chiefly noticeable in the stomach and upper portion of the intestinal canal, which are inflamed and disorganized, whilst "the large intestines are ordinarily filled with fæcal matters excessively hard;" and, as M. Tartra adds, "it is useful to remark that the urinary bladder does not contain urine, although the patients have not passed any urine." In two of the patients who came under the notice of this observer, one of whom lived twenty, and the other thirty hours after taking the poison, perforation of the stomach occurred. In the first of these cases (Obs. 10) the patient was a lad, aged sixteen years, in whom "the pulse became extremely small, the skin cold, the flow of urine was suspended, and the constipation resisted the use of lavements." In the other case (Obs. 14) the patient was a woman of the town, aged thirty-five years, in whom the symptoms were similar as regards the superficial coldness and imperceptible pulse, but the state of the urinary secretion is not mentioned. In the case of a painter, aged thirty-two years (Obs. 9), who died in nineteen hours from poisoning by nitric acid, M. Tartra informs us that "there escaped towards the end some drops of urine;" and it was remarked that whilst the "hideous aspect of the body of this unfortunate sufferer resembled that of a corpse," the mind was unimpaired. Lastly, in the case of a man, aged forty-five years (Obs. 8), who recovered from the immediate effects of the poison, but who died after a long interval from marasmus, the usual urgent symptoms, such as repeated vomiting, extreme cold-

[1] 'Gazette Médicale de Paris,' 2e sér., tom. v, pp. 188, 189, 1837.

[2] 'London Journal of Medicine,' vol. iii, p. 443, 1851.

[3] 'Traité de l'Empoisonnement par l'Acide Nitrique,' An. 10—1802.

ness of the surface, burning heat internally, and smallness of the pulse, were accompanied, M. Tartra informs us, by "absolute impossibility to pass urine," which from the history of the case appears to have been suppressed.

Poisonous and decomposing animal and vegetable matters have in like manner occasioned symptoms of collapse closely resembling those of cholera, if not the disease itself; but, as a rule, no attention has been paid to the urinary secretion in this class of cases. Dr. Christison[1] has remarked that "Game only decayed enough to please the palate of the epicure, has caused severe cholera in persons not accustomed to eat it in that state;" and similar cases have, some years since, come under my own observation, in which several members of the same family, after eating tainted mutton, suffered from cholera, three of whom died. On examination, it was found that the meat which had caused the mischief in this case, instead of being bad only on the surface, had undergone a rapid and peculiar decomposition near the bone. It is often, however, difficult, if not impossible, to recognise any morbid change in the animal matter to which this choleraic or pseudo-choleraic poisoning has been referred; and it is a curious fact that even milk itself, the secretion of which is commonly supposed to be altogether exempt from any choleraic influence, may in like manner undergo some unrecognisable change by which it acquires deleterious properties capable of producing symptoms of poisoning, which are sometimes analogous to and even identical with those of cholera. Among the more noticeable cases of this description is that which occurred in the year 1826 at Aurillac, a village in France, and which Professors Orfila and Marc were commissioned by the French Academy of Medicine to investigate. In this case fifteen or sixteen customers of a particular dealer in goat's milk were attacked on June 13th, with all the symptoms of violent cholera; and about twenty-four hours afterwards the goat too was taken ill with the same affection, and died in three days. Professors Orfila and Marc in their report stated that, in parallel cases which

[1] Op. cit., 4th edit., p. 635, 1845.

had been reported to them by the police of Paris, they had been unable to detect any mineral poison; that none of the received explanations were in their opinion satisfactory; and that they were disposed to ascribe the poisonous alteration of the milk to new principles formed by a vital process.[1] As an appendix to this case, it may be mentioned that on October 13th, 1856, about twenty persons in Malta were seized with violent vomiting and other symptoms of cholera, which were ascertained by the police physician, Dr. Felice Calleja, to be caused by drinking the milk of two goats, which was said to have acquired poisonous properties from the animals having fed on some wild herbs belonging to the genus oxalis or wood-sorrel.[2]

Among vegetable matters which are capable of producing injurious effects analogous to cholera are poisonous fungi, which in some cases produce narcotism, whilst in others the symptoms are those of gastro-intestinal irritation and collapse.[3] Tardieu[4] in his description of the symptoms produced by poisonous fungi, mentions that "the pulse is weak, the skin cold, and covered sometimes with violet-coloured blotches, and the urine suppressed;" death occurring in these cases "in from one to two or three days." He cites two cases in which a mother and daughter were poisoned by *amanita bulbosa;* the urine having been suppressed in the daughter from the time of the accident, whilst in the mother it was passed in the beginning of the illness, but subsequently her case like that of the daughter assumed the character of choleraic collapse. Flandin[5] has cited a case in which four persons in the same family were poisoned by eating a quantity of *amanita muscaria;* and M. Girard, in his report on this case, remarks, "We have found much analogy between the symptoms of this poisoning and those of

[1] 'Archiv. Gén. de Méd.,' tom. xv, pp. 460-462, 1827, cited by Dr. Christison, op. cit., 4th edit., p. 644, 1845.

[2] 'Medical Times and Gazette,' vol. xiii, new series, p. 456, 1856.

[3] Pereira, 'The Elements of Materia Medica and Therapeutics,' 4th edit., vol. ii, part i, p. 59, 1855.

[4] Op. cit., pp. 821-822, and 828-829, 1867.

[5] Op. cit., tom. iii, pp. 464-469, 1853.

Asiatic cholera. The alvine dejections have nearly always been white like rice-water and flaky." It is important moreover to notice that the analogy is strengthened by the post-mortem examination of the bodies in some of these cases, especially with reference to the condition of the blood, which has been observed to be "black and fluid."

There is little need to dwell on the analogy between cholera and the excessive action of drugs, for, like the poisons already referred to, it is chiefly those drugs whose normal action is limited to the gastro-intestinal mucous membrane which produce, when given in over-doses, effects similar to those of cholera. Among the more noticeable of these is croton oil, which in poisonous doses produces collapse closely analogous to that of cholera; but there is unfortunately no mention in the published records of such cases, either of the urinary secretion during life, or of the state of the bladder after death.[1] And similar omissions with respect both to the urinary secretion and the bladder occur in the records of other cases in which there has also been collapse resembling that of cholera; as, for example, in a case mentioned by Dr. Taylor,[2] in which a girl, aged eighteen years, was poisoned with castor-oil seeds, and whose appearance is described as "like one affected with malignant cholera;" and in the cases reported by Scot,[3] in which "three men having eaten, at the same time, the seeds of the coral plant, had all the symptoms of severe cholera."[4]

[1] 'Journal de Chimie Médicale,' tom. v, 2e sér., pp. 509-511, 1839; Orfila, op. cit., tom. ii, p. 108; Dr. Adam, 'Edinburgh Medical Journal,' vol. i, p. 932, 1855-6; and Dr. Greenhow, 'Medical Times and Gazette,' August 11th, 1866, p. 142.

[2] 'Principles and Practice of Medical Jurisprudence,' p. 266, 1865.

[3] 'Madras Report on Cholera,' p. xxxix, 1824.

[4] The evidence in favour of the excessive action in these cases being due to local irritation of the gastro-intestinal mucous membrane is confirmed by the observations of Pereira (op. cit., vol. ii, part i, pp. 401-402 and 406-407), 'On the Inhalation of the Dust of Euphorbium and of Croton Seeds.' In the latter case, owing to the local irritation being primarily limited to the pulmonary mucous membrane, several doses of castor oil had to be given before the bowels could be induced to act.

With reference to the various cases which have been as yet cited, it must be admitted that the closeness of the analogy is, at first sight, not altogether unfavorable to the theory which refers cholera to a specific blood-poison; and that it would consequently be allowable to assume, if the reasoning from analogy were limited to such cases, that the abdominal centre of the sympathetic nervous system may be only secondarily affected through the ramifications of the vaso-motory nerves. For such a theory of action might to some extent, though not altogether, be reconcileable with the irritant and corrosive effects produced on the gastro-intestinal mucous membrane, and which, in all of the preceding cases, were associated with, if they did not occasion and develop the collapse simulating that of cholera. But there remains to be considered a group of cases characterised by a closeness of analogy approaching in some respects almost to identity with cholera, in which it would not be allowable to assume that a blood-poison was the cause of the collapse. Well-marked illustrations of this occurrence of collapse, altogether independent of any possible connection with blood-poisoning, are to be met with in cases of perforation, rupture, and laceration of the stomach and small intestines; over distension of the stomach; and impassable obstruction in the small intestine. In all of these cases, with some others of an allied character, which will be incidentally noticed, there is usually a condition of collapse associated with suppression of urine, which not unfrequently has been, and, almost unavoidably, is very liable to be, mistaken for that of cholera; and if, as is sometimes alleged to be the case, such diagnostic errors are comparatively more often noticed in hospitals than elsewhere, it may be inferred that in private practice they are sometimes overlooked or concealed, and that in consequence the published records of their occurrence are incomplete.

The first cases of perforation of the stomach in which I noticed suppression of urine during life, with an empty and contracted state of the bladder after death, occurred before my attention had been specially directed to cholera; and

these earlier observations were subsequently embodied in a paper "On Perforating Ulcers of the Stomach," which was read at a meeting of the now extinct North London Medical Society in 1854. In this paper, of which an abstract was published in one or two medical journals, it was stated that "suppression of urine, although it does not appear to have attracted much notice, is as marked a symptom (in perforation) as it is in cholera, and that after death the urinary bladder is usually found to be empty and contracted." Among the cases occurring in my own practice which were cited on the occasion was that of a journeyman coach-painter, aged fifty-three years, in whom alarming hæmorrhage from the stomach had immediately preceded perforation; and although the patient survived nearly two days, no urine was passed, and the bladder after death was found empty and contracted. In the case of a Chelsea pensioner, aged fifty-nine years, employed as a potman, and in whom the immediate cause of perforation was a "dinner-treat" of roast pork and brandy, death occurred in thirty hours, during which time only six ounces of urine were passed, and after death the bladder was found empty and contracted. Whilst in the case of a bricklayer, aged sixty-six years, who died in sixteen hours and a half, and in the case of a retired publican, aged eighty-two years, who died in twenty-two hours, and whose case is of additional interest as one of the oldest on record,[1] there were no notes taken of the state of the urinary secretion during life, but in both of these cases, which occurred in the St. Marylebone Infirmary, the bladder after death was found empty and contracted.

Further inquiry in the same direction, and a more extended acquaintance with the literature of the subject, having convinced me that there is a very close relation between perforation and cholera, I endeavoured, in 1856, to prove in my work 'On the Nature of Cholera as a Guide to Treatment,'

[1] Dr. Brinton, "On Ulcer of the Stomach," 'Med.-Chir. Review,' vol. xvii, p. 171, 1856, states that the oldest case of perforation of the stomach he is acquainted with is that of a man who was, as in the above-mentioned case, aged 82 years.

that "the theory which ascribes cholera to an impression on the sympathetic nervous system, conveyed through the medium of the stomach or intestines," is very strongly supported by the analogical evidence of these and other allied cases which could be cited in its favour; and I proposed to include them with cholera, under the head of "Disorders of the Sympathetic Nervous System, depending on diminution or arrest of its functions, and produced chiefly through the medium of the stomach." There is no need for me to enter again into an exhaustive examination of the nature of this evidence, or to notice all the successive cases of mistaken identity, in which perforation and other morbid affections belonging to the same group have been from time to time confounded with cholera. In all of these cases there is essentially the same condition of collapse which is usually accompanied, as in cholera, with suppression of urine during life, and an empty and contracted state of the bladder after death; and although in a large number of the recorded cases of this description, including those which have and those which have not been mistaken for cholera, there is no allusion, after the perforation has occurred, to the secretion of urine during life, or the condition of the bladder after death; yet in all cases, so far as I can at present remember, in which I have sought and have been enabled to fill up this omission by subsequent inquiry, I have ascertained that suppression of urine, unaccompanied by any head-symptoms, is as well marked in such cases as it is in cholera. As an illustration of the extent to which this important condition in the analogy prevails, it may be mentioned that in the two cases of perforation simulating cholera, lately reported in the medical journals, and in neither of which is there any allusion to the urinary secretion or bladder, it has been ascertained on further inquiry that although in the St. George's Hospital case[1] of perforation of the stomach closely simulating but not apparently mistaken for cholera, no note was made of the state of the urinary secretion or bladder, and no information on the subject could be obtained; yet in

[1] 'Medical Press and Circular,' March 20th, 1867, pp. 271-272.

the other case[1] referred to, in which the patient died in the Cholera Ward of the London Hospital, after an illness of about two days and a half, from perforation of the duodenum near the pylorus, the acting resident medical officer who reported the case subsequently informed me by letter that there was "undoubted and continued suppression of urine." The additional information which was obtained in this latter case derives increased importance from the fact that in another case[2] of pseudo-choleraic collapse, which occurred in the same hospital only seven days after the last-mentioned case, and in a young female patient, who at the time had been an inmate of the hospital for more than a week on account of disease of the elbow-joint, the symptoms which led to her removal to and subsequent death in the Cholera Ward, were supposed to be indicative of "sudden cholera collapse;" her condition at the time being described as follows: "almost pulseless, quite cold, with general lividity of the surface, and blueness of the finger-nails." But it is important to notice in the history of this case, that whilst "several ounces of clear urine were drawn off," yet the patient was only semi-conscious. This difference in the symptoms, accompanying the collapse, of these two pseudo-choleraic cases in the London Hospital during the same month, admitted of being readily explained after death by the fact that the fatal collapse in the last case was due to Addison's disease, and that all the organs, with the exception of the supra-renal capsules, were found to be healthy; whilst in the other case, in which there was complete suppression of urine, the fatal collapse was the result of perforation of the duodenum near the pylorus.

So far as regards the literature of this subject, it must be acknowledged that the positive evidence in favour of suppression of urine during the collapse consequent on perforation is somewhat incomplete; for many of those who have published papers and cases descriptive of this lesion, have not apparently been aware of the significance of the symptom,

[1] 'Lancet,' Nov. 24th, 1866, p. 577.

[2] Ibid., March 9th, 1867, p. 299.

even when, as seems to have been more often than otherwise the case, it has not been altogether overlooked. Devergie[1] indeed in his valuable work on legal medicine distinctly states that in perforation of the stomach, the urine is suppressed; and in like manner, Valleix[2] has remarked "that the suppression of urine is sometimes very remarkable" in peritonitis from perforation; but there are very few writers of importance who have recognised that it is a usual, if not a constant symptom in such cases. Dr. Reeves[3] has indistinctly noticed that "the bladder is the organ most frequently affected" in perforation of the stomach; and that its implication is marked "by an incessant desire to pass water —a few drops only escaping;" and he has, moreover, remarked that "the bladder will be found on the introduction of a catheter, empty;" yet he refers to it merely as evidence of the peritoneal inflammation having become general, and does not connect it with shock to the abdominal sympathetic nervous system, although he has cited five cases of more or less sudden death from perforation of the stomach, and has further remarked that patients are sometimes liable to die suddenly in attempting to sit up after perforation has occurred. Dr. Stokes in his paper on "Peritonitis from Perforation of the Serous Membrane,"[4] does not even allude to suppression of urine in connection with perforation of the stomach; and in the abstract published of Mr. Leonard's paper on "Perforating Ulcer of the Stomach,"[5] which was read at the Medical Society of London in 1852, there is likewise no allusion to the urinary secretion, or to the state of the bladder. Dr. Crisp[6] has so far overlooked both the frequency

[1] 'Médecine Légale,' 3e édit., tom. iii, p. 776, 1852.

[2] 'Guide du Médecin Practicien,' 3e édit., tom. iii, p. 308, 1853.

[3] 'Diseases of the Stomach and Duodenum,' pp. 108, 109, 1856.

[4] 'Cyclopædia of Practical Medicine,' vol. iii, 1834.

[5] 'Medical Times,' vol. xxv, pp. 476, 477, 1852.

[6] 'Cases of Perforation of the Stomach from Simple Ulceration,' pp. 3-7, 1843. In one other case recorded by this author (case 4, pp. 10-12) there is indirect evidence of the urine being suppressed, as it is stated that the patient, a girl, æt. 15½, on the evening before her death "had a small quantity of gin and water, and about as much nitre as would lie on a sixpence."

and the importance of this symptom, as merely to state, without comment, that in the case of a woman who had died at 10.30 a.m., on December 27, 1837, from perforation of the stomach, no water had been passed "since 12 o'clock last night; bladder not distended;" and it was found after death that "the bladder was empty."

Among French writers to whom it will be useful to refer is M. Cazeneuve,[1] who has bestowed much attention on this diagnostic symptom, more especially in perforation of the small intestines, and who was at one time disposed to claim the merit of having been the first to draw attention to it in this latter class of cases. He relates a very impressive case of cancerous perforation of the stomach in a man, æt. 28, which came under his observation in the Military Hospital at Lille, in 1833. As no urine was passed, after the perforation in this case occurred, a catheter was introduced, but only two spoonfuls of urine were obtained, and it was found to be impossible to turn the catheter about in the bladder, owing to the very strong contraction of that organ. Subsequently the catheter was again passed, but no urine was obtained, and after death it was noted that the bladder was contracted and hidden behind the pubes. Laisné,[2] Gerard,[3] Lefevre,[4] and many other observers, who have written on this lesion, have generally failed to notice the state of the urinary secretion; although they have sometimes incidentally mentioned without comment, in what appears to have been the only case in which any note was made of this patho-

[1] 'Gazette Médicale de Paris,' 3e sér., tom. vi, p. 814, 1838.

[2] (Fr. Chaussier) 'Médecine Légale,' par MM. Lecieux, Renard, Laisné, et Rieux, 1819. A second edition of the Thesis on Perforation, translated by Dr. Weatherhead and dedicated, by permission, to Lord Eldon, was published in 1821.

[3] 'Des Perforations Spontanées de l'Estomac,' 1803. In a case cited by this author (Obs. 17, pp. 62, 63), in which a woman, aged 30 years, died from spontaneous perforation of the stomach, it may be inferred from the history of the case that there was suppression of urine during life; after death it was observed that "the bladder was very small."

[4] 'Archives Générales de Médecine,' 3e sér., tom. xiv, pp. 377-428, and tom. xv, pp. 28-59, 1842.

logical fact, that the urinary bladder after death was found contracted.

Among the isolated cases which have been recorded of perforation of the stomach, in which there is any reference to the urinary secretion and bladder, is a case, observed by Dr. (afterwards Sir Philip) Crampton,[1] of perforating ulcer of the stomach in a lady, æt. 29, in which death occurred from collapse twelve hours after the accident. The condition of the urinary secretion during life is not mentioned; but it is stated that after death "the urinary bladder was empty and contracted." In a case recorded by Dr. Carson,[2] of rupture of the stomach from rapid development of gas, which occurred in a sailor æt. 20, and who lived for fifteen hours, the condition of the urinary secretion after the occurrence of the accident is not referred to; but it was ascertained after death that "the bladder was contracted and empty." This case appears to have been somewhat complicated, as a slight trace of arsenic was detected in the stomach. M. Desgranges[3] has recorded the case of a young woman, æt. 18, who died in twenty hours from spontaneous perforation of the stomach, and in which the symptoms noted twelve hours before death were as follows:—"icy coldness of all the body, especially of the extremities; no pulse; eyes dull and hollow; temples sunk; face pale and cadaverous; and aphonia." There was desire but constant inability to pass urine, and later in the history of the case M. Desgranges refers to the fact of there being no urine. An instructive case of perforation mistaken for cholera has been recorded by Dr. Rigaud,[4] in which the patient died in twenty hours. Dr. Hérard[5] has recorded a case of perforation of the stomach in a woman, æt. 34, in which it was noted that the urine was suppressed. A female domestic servant, æt. 48, was admitted

[1] 'Transactions of the Association of Fellows and Licentiates of the King's and Queen's College of Physicians in Ireland,' vol. i, pp. 1-10, 1817.

[2] 'Edinburgh Medical and Surgical Journal,' vol. lxvi, pp. 27-31, 1846.

[3] 'Journal Général de Médicine,' 2e sér., tom. xv, pp. 145-164, 1821.

[4] 'Gazette Médicale de Paris,' 3e sér., tom. xxviii, p. 768, 1858.

[5] 'Bull. de la Soc. Méd. des Hôp. de Paris,' tom. iii, pp. 149-152, 1864.

into the Cholera Wards of St. Bartholomew's Hospital, Nov. 7, 1866, in whom, after death, there was found to be peritonitis from perforation of the stomach. Lastly, Dr. Barlow[1] has cited a case observed by Dr. Gull, of chronic ulcer of the stomach, with constriction of the pyloric extremity, in which there was excessive vomiting, and no urine was passed for forty-eight hours before death. In this case the bladder was found, after death, empty and contracted.

If from cases of perforation and injury of the stomach the attention be transferred to the corresponding class of cases affecting the small intestines, it will be found that suppression of urine is usually associated with the resulting collapse; and that in consequence of the common occurrence in such cases of this symptom, it has been referred to by some writers as diagnostic of the seat of mischief. There will not, however, be much difficulty in recognising, on further inquiry, that whether the mischief be the result of perforation or of obstruction of the small intestines, the suppression of urine which results from it must be regarded chiefly as an indication of the intensity of the collapse, consequent on the comparative suddenness of the mischief, and its nearness to the abdominal centre of the sympathetic nervous system; in the same way that a corresponding suppression occurs in severe cases of cholera, whilst in mild cases of this disease, which it has been customary to refer to as choleraic diarrhœa, there may be, and usually is, the characteristic cholera flux without any suppression of urine. If, for instance, the clinical history of those cases in which the mischief has been limited to one of the three divisions of the small intestines be carefully examined, it will be found that whilst suppression of urine is a very constant symptom in the collapse consequent on perforation or rupture of the duodenum, and also in corresponding cases affecting the jejunum, of which, however, there are but few examples on record, it is less constantly, although still very commonly observed in the strictly analogous cases in which the ileum has been the seat of mischief. With regard, moreover, to the comparative

[1] 'Medical Times and Gazette,' April 28th, 1866, p. 445.

value of this diagnostic symptom, it is important to notice that in a large proportion of the latter class of cases, in which the perforation has occurred in the course of typhoid fever, there has been diminution of the urinary secretion as a consequence of the fever, previous to and apart from any severe affection or injury of the ileum, and that this diminution, being associated with the continued formation and accumulation in the system of the urinary constituents, is pathologically a distinct condition from the diminution and suppression which occurs in those cases which are analogous to cholera, and in which, as in cholera, the failure of excrementitious elimination is an index of the extent to which the sympathetic nervous system is affected during collapse.

The following illustrative cases referrible to the duodenum may be cited in favour of these remarks:—1. The case already mentioned of spontaneous perforation of the duodenum, three lines from the pylorus, which was lately admitted into the Cholera Ward of the London Hospital, and in which there was "undoubted and continued suppression of urine." 2. A case, reported by M. Petrequin,[1] of spontaneous perforation of the duodenum, eight lines from the pylorus, in which the collapse very closely resembled that of cholera, for there was suppression of urine, cyanosis, the Hippocratic face, and the pulse was imperceptible at the wrist. The state of the bladder after death was not noted. 3. A case, reported by Dr. Humphrey Sandwith,[2] of perforation of the middle of the duodenum by a worm, in which the pulse at times was scarcely perceptible at the wrist, and when a catheter was passed only a few ounces of urine were withdrawn. After death the bladder was found empty and contracted. 4. A case, reported by Dr. Symes,[3] of laceration of the duodenum, from external violence, which the patient survived only twenty hours. In this case, which differs somewhat in character from the preceding, as the injury occurred from without inwards, there was a constant desire

[1] 'Archiv. Gén. de Méd.,' 2e sér., tom. xii, pp. 483-485, 1836.

[2] 'British Medical Journal,' March 9th, 1861, pp. 249, 250.

[3] 'Dublin Quarterly Journal,' vol. xlii, pp. 497-498, 1866.

to pass water, but it is not stated that any was passed. After death the bladder was found empty and contracted. 5. A case, recorded by Prof. Rembold,[1] of strangulation of the first portion of the duodenum, in which, on passing a catheter, only some drops of urine were obtained. 6. A case of perforation of the duodenum in a clerk, aged thirty years, recorded by M. Oulmont,[2] in which there was suppression of urine with vesical tenesmus; and when, owing to the patient believing that the bladder was about to burst, a catheter was passed, not a drop of urine could be obtained. After death the bladder was found strongly contracted and quite empty. In this case there was for some hours before death persistent delirium, which appears, however, to have been due, not to the suppression of urine, but to the patient's previous habits of excessive drunkenness, which had led to occasional attacks of insanity, during which he had thought one day to strangle his child, and on another occasion to kill his wife. Lastly, in a small series of cases of duodenal perforation, recently published by Dr. Andrew Clarke,[3] there is, beside the case already referred to as having died in the Cholera Ward of the London Hospital, a well-marked case (Obs. 2), of a lad, aged nineteen years, who was admitted into the London Hospital "in a state of partial collapse, and alleged to be suffering from retention of urine." By means of a catheter, easily introduced, the bladder was found empty. After death it was ascertained that the perforation of the duodenum had occurred about one inch and a half from the pylorus. "The right cavities of the heart were gorged with blood; the left empty." Dr. Clarke, in commenting on this case, remarks that "one could scarcely have wondered if it had been mistaken for cholera." In the case of a watchmaker, aged twenty years, which is the third case in this series, the urinary secretion and bladder are not referred to, but the appearance of the patient is described as "like that

[1] 'Gazette Hebdomadaire,' 28 Avril, 1865, pp. 271, 272.

[2] 'Archives Médicales de Strasbourg,' Août, 1836; cited by M. Forget, 'Gazette Médicale de Paris,' 2e sér., tom. v, p. 229, 1837.

[3] 'British Medical Journal,' June, 1867.

of a choleraic in collapse."[1] There are two other cases of perforation of the duodenum recorded by Dr. Clarke, which it may be useful to notice in connection with this subject, as they illustrate the relatively milder effect of a slight and gradual leakage compared with a more sudden and extensive escape of the duodenal contents. In one of these cases the patient was a man, aged thirty-five years, who died from perforation of the duodenum, which was situated about half an inch from the pylorus. When first seen, only a slight leakage had apparently occurred; the extremities were warm, and some healthy urine had, a short time previously, been passed. Six hours before death there was undoubted evidence of peritonitis from perforation, with the characteristic collapse. The perforation of the duodenum in this case was ascertained to be about half an inch from the pylorus; but the state of the bladder after death is not mentioned. In the other case (Obs. 6), which is of a somewhat doubtful character, a man, aged forty-three years, was supposed to have had a slight leakage from duodenal perforation, and it is stated that the urine was normal; but the diagnosis in this case could not be verified, as the patient recovered.[2]

Cases of perforation, of obstruction, or even of traumatic lesion of the jejunum, are, as already stated, rare; but among the few which have been recorded, there is one of considerable interest observed by M. Cazeneuve,[3] in which a man, æt. 24, suffered from two attacks of peritonitis from

[1] Corresponding cases have been published by M. Poisson ('Bull. de la Soc. Anat. de Paris,' tom. xxx, pp. 300, 301, 1855;) by Mr. Stilwell ('Lancet,' July 18th, 1846, p. 67); and by Sir David J. H. Dickson ('Med.-Chir. Review,' vol. xxxiii, pp. 586, 587, 1840.)

[2] In connection with these cases it may be useful to notice that acute ulceration mostly limited to the first portion of the duodenum, and to which Mr. Curling ('Med.-Chir. Trans.,' vol. xxv, 1842) has directed attention as a not unfrequent sequel to burns, especially in young subjects, appears to be an occasional consequence of what at present can only be vaguely referred to as shock, which appears to act by causing an arrest of nutrition, and which would therefore be strictly allied to, though differing in degree from, what occurs in cholera and in some of its analogies.

[3] 'Gazette Médicale de Paris,' 3e sér., tom. vi, obs. 2, p. 813, 1838.

perforation, which occurred in January and in April of the same year. It is mentioned in the report of this case that there had been, at the time of the first attack in January, vesical tenesmus with difficulty in the urinary secretion: whilst in the fatal attack in April there had been suppression of urine. After death, perforation of the ileum near the ilio-cæcal valve, and of the jejunum near its middle were found; and the bladder was empty and hid behind the pubes. Dr. Barlow[1] has recorded a case of fatal obstruction of the jejunum, in which there was complete suppression of urine for six days; a catheter having been passed and no urine obtained. And a case of traumatic rupture of the jejunum in a labourer, æt. 31, which was followed by death in forty hours, occurred lately in the practice of Mr. Curling[2] at the London Hospital. In this case the collapse closely resembled that of cholera; there was intense pain in the hypogastric region; vomiting; coldness of the extremities and also of the tip of the nose; pulse imperceptible at the wrist, and very feeble in the brachial artery; and cyanosis. There is no reference, in the report of the case, to the state of the urinary secretion during life, but after death the bladder was found empty and contracted.

Although it must be admitted that comparatively little attention has been directed to the state of the urinary secretion in perforation or in other lesions of the stomach, duodenum, and jejunum, yet there has been great and, in some respects, undue importance assigned to it in those cases in which the ileum has been affected; and whilst some distinguished writers have referred to it as in some degree diagnostic of perforation, others have regarded it as an almost certain indication of obstruction in the small intestine, chiefly at this part. Among the former may be cited M. Cazeneuve,[3] who has recorded three cases of perforation of the ileum (Obs. 1, 2, and 5) occurring in the course of typhoid fever, in which

[1] 'Guy's Hospital Reports,' vol. ii, pp. 378-380, 1844; and 'Medical Times and Gazette,' April 28th, 1866, p. 444.

[2] 'London Hospital Reports,' vol. ii, pp. 287-288, 1865.

[3] 'Gazette Médicale de Paris,' 2e sér., tom. v, pp. 818-821, 1837.

there was more or less vesical tenesmus, with suppression of urine; and in each of these three cases the bladder was found empty and contracted. Subsequently, however, M. Cazeneuve[1] has remarked that "suppression of urine has not occurred in all cases; often, also, it is not complete. I should only have (he adds), in order to prove this, to cite the silence of authors on this point. In effect, it is not mentioned by M. Andral, Louis, Chomel. M. Broussais cites only two or three examples of it; moreover, cases have been related in which the urinary secretion has continued." Among those who have bestowed special attention on this symptom is M. Judas,[2] who, when Assistant Physician to the Gros-Caillou Military Hospital at Paris, recorded two cases of perforation of the ileum which occurred in that hospital, and in both of which there was suppression of urine. In one of these cases, that of a soldier aged twenty-three years, a catheter was passed without, however, obtaining a drop of urine; and after death the bladder was found empty and very much contracted. In the other case, when a catheter was passed, it could not be depressed, and only a few drops of clear and inodorous urine were withdrawn: the bladder, after death in this case, was found empty and contracted to the size of a pigeon's egg. In a case which occurred in the practice of M. Piorry,[3] in which a man, aged thirty-one years, had perforation of the ileum in the course of typhoid fever, there was for some time suppression of urine as a result of the perforation, but subsequently, during the last few days of life, the urine was again secreted and passed; showing that the suppression in these cases is not so much due to either the fever or the inflammation, as to the shock consequent on the perforation. Whilst in cases unconnected with typhoid fever, as, for example, in those produced by a worm, or by external injury, suppression of urine, with an empty and contracted state of the bladder after death, has been noted by Mr. Kell,[4]

[1] 'Gazette Médicale de Paris,' 3e sér., tom. vi, p. 814, 1838.
[2] Ibid., pp. 722, 723.
[3] Ibid., pp. 44, 45.
[4] 'London Medical Gazette,' vol. ii, pp. 649-650, 1828.

by M. Andral,[1] by M. Baraduc,[2] and by some other observers.

But far more importance has been assigned to suppression of urine in the collapse which results from intestinal obstruction than, perhaps, in any other class of cases; and the following case of strangulation of the lower part of the ileum, which has been lately recorded by M. Dexpers,[3] affords a good illustration of the analogy between such cases and cholera. The patient was a soldier, who, when brought to the hospital, presented, we are informed, the appearance of a patient suffering from cholera; his eyes were sunk; his complexion cyanosed; his hands blue and cold; the skin wrinkled; and the pulse small, hard, and very frequent. The succeeding night was passed in mortal agony; in spite of frequent desires, it was impossible for him to pass either fæces or urine, and the introduction of a catheter brought away only a few drops of urine. The intellectual faculties were unimpaired. After death the bladder was observed to be empty and completely hid behind the pubes. Dr. Besnier,[4] in a valuable memoir on intestinal obstruction, has recorded a fatal case (Obs. 128) of strangulation of the small intestine in a woman, aged forty-seven years, who, "in consequence of a bath too hot taken immediately after having eaten, was suddenly seized with a very sharp and sudden pain in the abdomen." There was the usual collapse with constipation and suppression of urine from that time, and it was ascertained on the day after her admission that the bladder was empty. And Mr. Robinson[5] has recorded the case of a woman, aged sixty-three years, who died in thirteen hours from collapse with suppression of urine, which was supposed to be cholera, but after death it was ascertained that there was strangulation of the ileum. With reference to such cases it is curious to notice

[1] 'Clinique Médicale,' tom. ii, 3e édit., pp. 602-604, 1834.

[2] 'Bull. de la Soc. Anat. de Paris,' tom. xiii, pp. 336-340, 1838.

[3] 'Recueil de Mémoires de Médecine, de Chirurgie, et de Pharmacie Militaires,' tom. xviii, 3e sér., pp. 195, 196, 1867.

[4] 'Des Etranglements Internes de l'Intestin,' Paris, pp. 194-196, 1860.

[5] 'London Journal of Medicine,' vol. iii, pp. 443-446, 1851.

the great difference of opinion which has prevailed respecting the importance and the meaning of this suppression of urine; for whilst, on the one side, the late Dr. Barlow,[1] in common with some other observers, alleged that it might be considered diagnostic of that form of constipation which results from obstruction of the bowels situated high up, and that when the obstruction is low down then the urine is not suppressed in these cases of obstinate constipation, owing, as was erroneously supposed, to a sufficient amount of fluid for carrying on the urinary secretion being allowed to enter the system in the latter class of cases, but not in the former; on the other side it will be found that a different explanation of the fact has been given in the lately published and posthumous work of Dr. Brinton[2] on intestinal obstruction, in which the diagnostic value attached to this symptom by Dr. Barlow is altogether denied, and Dr. Brinton, in admitting the frequent occurrence in these cases of suppression of urine, expresses his belief that "the amount of vomiting is the truest symptomatic correlative of the diminution of the urine," and consequently agrees to some extent with Dr. Besnier,[3] who, in directing attention to the opinions on this subject of "English physicians," remarks, that the state of the urinary secretion, although "useful to consult, nevertheless does not furnish any indication which has an absolute value;" and that to enable us to infer something from the small quantity of urine secreted there must be neither peritoneal inflammation nor any considerable or frequent vomiting; "for these conditions (he adds) are sufficient to bring about diminution or even suppression of the urinary secretion." It may here, perhaps, be allowable to remark that any correlation which may exist between vomiting and suppression of urine in these cases appears to be simply the result of both conditions being primarily dependent on the affection of the abdominal

[1] 'Guy's Hospital Reports,' 1844; and 'Medical Times and Gazette,' April 28th, 1866.

[2] 'Intestinal Obstruction,' by William Brinton, M.D., F.R.S., edited by Thomas Buzzard, M.D., London, 1867.

[3] Op. cit., pp. 305, 306.

sympathetic nervous system; and this seems to have been subsequently, though very imperfectly, recognised by Dr. Brinton, who refers with evident hesitation and doubt to the connection between this form of suppression of urine and what he terms "mere collapse."

The opinions which have been expressed by the above-cited and other authorities respecting the diagnostic value of suppression of urine in such cases as the preceding appear to be all defective, in so far that the suppression is alleged to be connected with or dependent on something which is not essential to its occurrence, although it may be, and perhaps often is, associated with it. Hence the importance of carefully collating the clinical history of all those cases in which, as in cholera, it characterises the collapse; for by thus extending the range of the inquiry it will be conclusively proved that this symptom may occur in corresponding conditions of collapse, independent, for example, of any one of those four cardinal phenomena, of pain, vomiting, intestinal obstruction, and intestinal flux, which have each been referred to by distinguished writers on the subject as its correlative, since by means of such extended inquiry it can be clearly demonstrated that suppression of urine is not necessarily dependent on any of these associated conditions. This will become more and more evident as the inquiry proceeds, but it will be convenient on this occasion to remark that its occasionally more or less complete independence of pain is fully proved by the fact that there may be epigastric pain quite equal in intensity to that of cholera, or any analogous disease, without any suppression of urine; that cases of painless choleraic diarrhœa have been observed in which the urine has been suppressed; and that in cases of cholera itself the suppression is often maintained for a long time after all pain has ceased in patients who ultimately recover. Whilst its alleged dependence, either on intestinal obstruction or on flux from the stomach or bowels, is in like manner fully disproved by cases in which neither of these two apparently opposite conditions has been observed, the term apparently being here introduced in consequence of these two conditions,

so far as the suppression of urine is concerned, admitting of being reconciled. For the suppression of urine in these cases is supposed to represent simply a want of water, and this deficiency is alleged to be consequent on the supply being so completely shut off in cases of intestinal obstruction, and so completely drawn off in cases of intestinal flux, as to reduce the remaining fluid in the body very considerably below what is required to carry off the excrementitious matters usually eliminated by the kidneys. Ample evidence of a very conclusive kind could be cited in opposition to this theory of the suppression of urine during collapse being due to a reduced supply of fluid; but it will be sufficient for the present to quote the two following cases to show that the urine may be suppressed during collapse from structural lesions in the immediate neighbourhood of the stomach, without the integrity of the gastro-intestinal mucous membrane being impaired. In the first of these illustrations, which has been recorded by Dr. McSwiney,[1] a man, aged forty-five years, died from collapse with suppression of urine, fifty-eight hours after the spontaneous rupture of the common bile-duct. The lesion, which occurred about three quarters of an inch from the entrance of the duct into the duodenum, was probably connected with over-distension of the gall-bladder from starvation; and although the lower portion of the ileum in this case was much diminished in calibre, it was pervious to the passage of fluid. In the second illustration, which was observed by Dr. Wolf,[2] of Bonn, there was a corresponding rupture of the hepatic duct in a woman, aged sixty years, who died twenty-four hours after the occurrence of this lesion. The resulting collapse in this case closely resembled that of cholera, and only a few drops of urine were discharged. The urine has in like manner been sometimes observed to be suppressed in severe abdominal operations, and among the more distinguished observers who have specially noted this fact is M. Claude-Bernard,[3] who informs us that in the operations

[1] 'Dublin Quarterly Journal,' Nov., 1866, pp. 518, 519.

[2] 'Journal des Progrès des Sciences et Institutions Médicales,' tom. xiv, pp. 245, 246, 1829; cited from 'Journal von Graafe,' 12e, B. S., 370.

[3] 'Comptes Rendus,' tom. xlvi, p. 161, note, 1858.

required to expose the renal vein, in which it is necessary to open largely the abdomen, there is "nearly always (as a consequence of the operation) in the dog and in the rabbit, if not immediately, at least after a few moments, suppression of urine."

The importance which has been attached to suppression of urine in cases of collapse simulating that of cholera must not, however, lead to other points in the analogy being undervalued or overlooked; for in some recorded cases belonging to the preceding group, in which there is no mention of the urinary secretion or bladder, much useful information on some other facts in pathology may not infrequently be obtained. With reference more especially to the condition of the vascular system, it should be observed that there is often a very close resemblance between these cases and cholera, not only as regards the frequent occurrence of cyanosis, and the failure, often for several hours before death, of the pulse at the wrist, which have been noted in many of the cases already cited, as well as in others in which there is no mention of the urinary secretion; but even the physical condition of the blood and the spasmodic contraction of the blood-vessels have occasionally been observed to be also similar. In a case, for example, of spontaneous perforation of the stomach in a girl, aged twenty-one years, observed by M. Ubersaal,[1] Senior Physician to the Orphan Asylum at Strasburg (a preparation of which has been preserved in the Museum of the Faculty of Medicine), it was found, on bleeding the patient from the arm to the extent of eight ounces, that the blood drawn from the vein was so thick as to flow with difficulty; and although the collapse in this case was not at first so well marked as it usually is after perforation has occurred, yet only a small quantity of urine was passed. The state of the bladder after death was not noticed. And Dr. Ebermaier,[2] in an ably written article on perforation of the stomach, states that in two of the cases which he has cited (cases 2 and 8) the patients were ordered

[1] 'Archiv. Gén. de Médecine,' tom. xxvi, pp. 412-414, 1831.

[2] 'Journal Complémentaire du Dictionnaire des Sciences Médicales,' tom. xxxi, pp. 34 and 164, 1828.

to be bled, and that in each case the attempt to obtain blood from a vein failed.

Another point in the analogy between these cases and cholera, to which it may be useful to refer, is the spasmodic contraction of the muscles, which has been specially noticed by M. Gerard[1] in a well-marked case of spontaneous perforation of the stomach in a man, from twenty-eight to thirty years of age, in whom "the muscles of the abdomen were in a state of such violent contraction that the anterior wall of this cavity appeared glued to the vertebral column, and offered the hardness of a board." The patient was ordered to be bled, in accordance with the usual practice at that time, and it was noticed that "as soon as the lancet was withdrawn from the vessel the blood spurted out more than ten feet, and immediately stopped, without the possibility of the surgeon, who was very dexterous, being able to obtain more." M. Gerard, in commenting on this case, informs us that these violent contractions are not an infrequent symptom also in wounds of the stomach, and he has cited a remarkable instance which occurred in the military hospital at Strasburg, into which a soldier had been admitted apparently dead from a large wound in the abdomen below the zyphoid cartilage, and through which the contents of the stomach escaped. The patient rallied, but died the next morning, after suffering great pain; and in dissecting the body it was found that in the muscles of the arm, but especially in those of the thighs, an innumerable number of the muscular fibres were broken. In like manner it has been remarked by Orfila,[2] and other observers, that spasmodic rigidity of the muscles during life is "among the lesions of tissue produced by arsenious acid."

Well-marked pulmonary collapse, which has been already noticed in connection with irritant poisoning, was observed by Dr. Cotton[3] in a case of large perforating ulcer of the stomach communicating with the transverse colon. The

[1] Op. cit., pp. 1-4, 1803.

[2] Op. cit., tom. i, 4e édit., p. 344, 1843.

[3] 'Medical Times and Gazette,' Jan. 12, 1856, pp. 38, 39.

patient in this case was a shoemaker, aged forty years, who died in the Brompton Hospital, eighteen days after fæcal vomiting had occurred, from "exhaustion or collapse;" and the lungs, which were found after death to be free from tubercle, were "very much collapsed." In connection, moreover, with pulmonary collapse it may be remarked that an alteration characterised by diminution or absence of the voice, such as is common in cholera, is also very noticeable in many of these cases of analogous collapse, and it has been specially referred to by Dr. Murchison[1] in his elaborate paper on gastro-colic fistula.

In directing attention to these and other accompanying conditions in collapse, it is necessary at the same time to state that even if they could all be detected and analysed with the same facility as suppression of urine, which, generally speaking, is not possible, yet none of them indicates either so clearly or so exactly the character and the extent of that general affection of the system which, in the case of cholera and its analogies, has been inferred to result from a central arrest of organic function. Failure of the circulation, cyanosis, and superficial coldness, are none of them so distinctive as suppression of urine is of such an arrest of organic function, for they may to some extent occur without it. In like manner, the pulmonary function also fails to supply any corresponding amount of information; for even if, in relation to organic life, carbonic acid could reasonably be assumed to be the correlative of urea, yet there is not the same facility for ascertaining either the extent to which its development has been arrested or the extent to which it has been unduly retained in the blood. With reference, therefore, to the alleged connection between the lungs and cholera through the medium of the blood, to which great and apparently undue importance has sometimes been attached, although there is evidence to show that the development of carbonic acid during intense collapse is more or less completely arrested, and that consequently the air inhaled only partially enters the lungs, and is expired almost, if not occasionally

[1] 'Edinburgh (Monthly) Medical Journal,' vol. iii, p. 132, 1858.

altogether, unchanged, still it must be admitted that the lungs can never, like the urinary bladder, be empty and contracted for an equally lengthened period of life, nor can the formation of carbonic acid, like that of urea, be so completely interrupted for the same time. It may consequently be affirmed that the state of the urinary secretion is the best guide that could be selected in investigating the nature of choleraic and allied forms of collapse, the assistance afforded by it being twofold, and derived partly from direct observation and partly from analogy. With respect to the former, it has been satisfactorily demonstrated by the researches of Dr. Garrod[1] and other able investigators[2] of the chemical pathology of cholera, that the formation of urea is checked or suspended during choleraic collapse; and although there are no corresponding observations on the suppression of urine in analogous cases of collapse, and consequently no direct authority for stating that the suppression in such cases is due to a like arrest of organic function, yet it must be admitted that the presumptive evidence is very great, if not almost conclusive, in its favour. For unless we are prepared to admit that the suppression of urine in these cases of analogous collapse is connected with the arrested formation of urea, it would be impossible to avoid coming in contact with one of two dangerous fallacies, which represent Scylla and Charybdis in the pathology of collapse. For if, in these cases, urea is uninterruptedly formed during collapse, it must be either discharged or retained; in the one case we should have to face the insurmountable obstacle of vicarious secretion, insurmountable at least on this occasion, because it would be necessary to assume that the amount of fluid might be reduced below that required for renal secretion, and yet suffice, not only for a distant and extra-visceral secretion like that from the mammary gland, but also for an adjoining and vicarious secretion; whilst in the other case we should have

[1] "On the Pathological Condition of the Blood in Cholera," 'London Journal of Medicine,' 1849.

[2] Dr. O'Shaughnessy 'On the Chemical Pathology of Malignant Cholera,' p. 61, 1832.

to admit that urea might accumulate in the blood during collapse without causing mischief, although all past experience and all direct observation on the subject lead to the inference that uræmic poisoning would follow with unusual rapidity the accumulation of urea in blood which, although too inspissated, as it is alleged, for renal secretion to occur, is yet not so inspissated as to arrest the mammary secretion or to impede the circulation through the brain.

Apart from analogy it would, perhaps, be scarcely possible to estimate correctly the value of this symptom as indicative of collapse, for it is still very generally supposed that the suppression of urine, if not the collapse itself, is essentially dependent in cases of cholera on the flux from the stomach and bowels; and Dr. Copland,[1] who agrees on this point with Sir Thomas Watson,[2] Dr. Wood,[3] of Philadelphia, and other distinguished writers, may be said to represent very fairly the prevailing opinion when, in his latest publication on the subject, he states without reserve that "the ischuria (in cholera) is a consequence of the excrementitial fluid of the blood being all discharged by the digestive canal and skin, none being left that can be removed by the kidneys." In reply to this assertion it will be sufficient to mention that, not only is there ample evidence derived from direct observation to refute it, in addition to the evidence which has been adduced from analogy, but that there is, moreover, a self-destructive fallacy in thus supposing that the digestive canal could serve for the complete escape of excrementitial matter usually discharged by another outlet, whilst the elimination of excrementitial matter proper to the canal itself is altogether checked. For just in the same way that jaundice results from the increasing accumulation of the essential elements

[1] 'Dictionary of Practical Medicine,' vol. iii, part ii, p. 1006, 1858.

[2] 'Lectures on the Principles and Practice of Physic,' 4th edit., vol. ii, p. 642, 1857.

[3] "In epidemic cholera, in which the urinary secretion is sometimes entirely arrested, the brain remains remarkably clear, in consequence, no doubt, of the copious discharges from the skin and alimentary mucous membrane."—'A Treatise on the Practice of Medicine,' 4th edit., vol. ii, p. 599, 1855.

of bile in the blood, and uræmic poisoning from the increasing accumulation in like manner of urea, so the absence of these two morbid conditions shows that during collapse there is no excess of excrementitial matter for either the kidneys or the liver to eliminate, and therefore no urine is conveyed to the bladder and no bile to the alimentary canal. It is, indeed, chiefly in consequence of this parallel arrest of the organic functions presided over by the abdominal sympathetic nervous system that the primary seat of the disease in cholera, as in analogous diseases, has been referred to the digestive canal, for no corresponding arrest of function usually occurs in those organs which are remote from this nervous influence; and the analogy which has been traced in the preceding groups of cases tends, without exception, to this conclusion. But before any just estimate can be formed of the value of analogy in cholera, it is necessary that preconceived opinions in favour of a lung theory of the disease should be given up, so as to allow the attention for a time to be transferred from the outworks to the centre of organic life, for otherwise it would be impossible to give any physiological explanation of the fact that lactation continues and the urine is suppressed, whilst the condition of the brain itself is undisturbed; and although analogy can only be referred to for the purpose of supplying indirect evidence, yet, on the present occasion, this is so strongly in favour of cholera being primarily due to an affection of the sympathetic nervous system, developed through the medium of the digestive canal, as scarcely to need any further evidence to support it. It may, however, before concluding, be useful to state that such experimental evidence as the subject admits of strengthens the argument from analogy; and although evidence of this kind, when based on a protracted and complicated operation, is usually very questionable, there is some which appears to be free from any such objection, as, for example, Dr. Davey's [1]

[1] 'The Ganglionic Nervous System,' p. 196, 1858. The pressure of an aneurism, by interfering with the nervous supply to the small intestine, appears to have caused "excessive diarrhœa" in Dr. J. W. Ogle's case of aneurism of the superior mesenteric artery at St. George's Hospital. ('Path. Trans.,' vol. viii, pp. 168-170, 1857.)

important observation, that "the fatal depression in cholera, consisting in the complete annihilation of the action of all the vital organs, may be at any time simulated by pressing the solar ganglion on the fore part of the bodies of the vertebræ over which it lies."

P.S.—Since the above was written some important evidence in support of the argument contained in this paper has been supplied by M. Moreau's experiments on section of the abdominal nerves in relation to intestinal flux. ('Comptes Rendus des Séances de l'Académie des Sciences,' tom. lxvi, pp. 554-557, 1868; cited in 'Medical Times and Gazette,' April 11, 1868, p. 397.)

ON

EXCESS OF UREA IN THE URINE

AS A GUIDE TO THE

DIAGNOSIS AND TREATMENT OF CERTAIN FORMS OF DYSPEPSIA AND NERVOUSNESS.

BY

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IN the year 1864 I had occasion to examine the urine of a patient who was suffering from symptoms of a nervous and dyspeptic character. The urine was frothy, somewhat pale and clear, sp. gr. 1025; it had an acid reaction and a strong urinous smell; it contained neither sugar nor albumen, and only a few small crystals of oxalate of lime were visible under the microscope, but the addition of nitric acid caused intense effervescence, and about twenty minutes after this had subsided crystals of nitrate of urea began to form and ultimately filled the interior of the test-tube. During a period of rather more than three weeks I examined this patient's urine every second day, and invariably found it to contain urea in quantity sufficient to crystallize readily on the addition of nitric acid, without any previous evaporation or concentration of

the urine. The degree of crystallization varied at each examination, in consequence, doubtless, of a variation in the amount of the urea contained in a given bulk of the urine; but inasmuch as I could not persuade the patient to save and measure the quantity of urine voided in twenty-four hours, I am unable to state whether less urea was secreted when the crystallization was at its minimum, or whether the variation arose from an increase in the quantity of water and a consequent dilution of the urea. The patient, however, declared, as the result of his observation, that he was passing about two pints of urine, and that the quantity scarcely varied from day to day.

The patient was thirty-seven years of age, of spare habit, in good condition, and apparently healthy; he was not a large eater, but he relished his food, and ate meat three times a day. His principal complaint was of nervousness and depression of spirits, indisposition for exertion, whether mental or bodily, and dyspepsia, with irregularity of the bowels, acidity of stomach, and excessive flatus.

The case made a strong impression on me at the time, for the reason that I was not prepared for the discovery of so much urea, and had never heard such cases spoken of by my professional friends or seen any notice of them by authors. I have therefore kept the matter steadily in view, and within the last two years have examined the urine of every patient in whom, judging from the character of the symptoms, I have thought it likely that urea might exist in excess.[1] The result has been that I have detected urea in

[1] By excess of urea in the urine I mean simply to imply that there is an excess of urea in a given bulk of the urine—an excess sufficient to lead to its crystallization on the addition of nitric acid without any previous concentration of the urine. In some of the instances referred to it was ascertained by actual analysis that *the quantity of urea excreted in twenty-four hours was decidedly above the average*, and I believe that it was so in every instance; but inasmuch as considerably difficulty is experienced in private practice in obtaining the whole quantity of urine voided in twenty-four hours, the amount of urea excreted in that period has been determined only in six cases, and I am therefore unable to speak positively as to the fact in the other instances.

excess in twenty-seven cases, and so well marked and peculiar are the symptoms by which that condition of the urine is accompanied, that now, if the features of the patient's malady induce me to suspect its existence and to examine the urine with a view to its discovery, I am seldom disappointed in obtaining the characteristic crystals of nitrate of urea.

The cases under discussion resemble each other so closely in many of their more prominent symptoms, that it will suffice to give the details of one or two which may be fairly taken as samples of the whole.

A. T—, æt. 43, a spare but healthy looking man, 5 feet 8 inches in height, 10 stones 9 lbs. in weight, of florid complexion, but somewhat bilious temperament, reported that he had been nervous throughout his life, and since the age of thirty had not felt well for any lengthened period. If he had unusual worry in his profession or any anxiety in his family, or even if he was unable to obtain his ordinary amount of outdoor exercise, he was apt to become irritable, low, and depressed, indisposed for exertion, and easily fatigued, unable to fix his mind on any subject requiring continuous application, and afraid to encounter the rubs and vexations incident to his daily life. His present illness he attributed to anxiety arising from the illness of one of his children, together with the want of his usual exercise. He had been remarkably well for some time previously, and became conscious of some change or disturbance in his system within a week after the commencement of his trouble. At first he became restless at night; then drowsy after meals, especially after dinner, and began to suffer from flatulence and acidity; shortly he found himself nervous and dejected without obvious cause, especially in the morning, unable to apply himself to his professional duties, and unwilling, if not unequal, to take his wonted exercise. Nervous fancies and wretched feelings, occasional giddiness, fugitive pains in various parts of the body, and a sense of extreme languor and weariness, rendered his life miserable. For six weeks before he consulted me he

had been taking a series of Turkish baths, and had dieted himself carefully, but notwithstanding his care the dyspeptic symptoms had continued, and his mental disquietude was more constant than before.

When I first saw him he was restless at night, but obtained a fair amount of unrefreshing sleep; his skin was rather dry, but perspired readily after exercise; his pulse ranged from 84 to 96; his appetite was tolerable, but not so good as usual; he ate meat twice a day, and was in the habit of drinking four or five glasses of sherry or an equivalent amount of claret, but he never drank beer; after each meal he suffered greatly from flatulence and drowsiness, and after breakfast experienced a sense of uneasiness about the head and of heat and weariness of the eyes. His bowels were somewhat torpid, but usually acted without the aid of medicine; his urine he said was "all right," and with the exception that it was frothy or strongly beaded it certainly did not present an unhealthy appearance. Occasionally it threw down a deposit of the lithates, but more commonly it was clear, pale, acid, of a strong urinous odour, sp. gr. 1026, and was free from sugar and albumen. Under the microscope a few octohedral crystals of oxalate of lime and here and there a few epithelial cells were visible, but they were scarcely more numerous than in healthy urine. On the addition of nitric acid to the extent of about one third of the bulk of the urine a strong effervescence took place, and within twenty minutes after this had subsided crystals of nitrate of urea had formed to such an extent that the whole of the urine in the test-tube appeared to be converted into a solid mass. On some occasions the crystals of nitrate of urea did not occupy above half the bulk of the urine, but commonly they filled two thirds of the space occupied by the urine. My friend Dr. Dickinson, who kindly examined a sample taken from the entire quantity of the urine (1927 c.c.) voided in twenty-four hours, reported that the day's excretion contained 53·9 grammes of urea, or nearly 21 grammes in excess of the average amount excreted within that period according to the observations of Dr. Parkes.

The next and only other case which I shall detail is of a very similar character. The patient, a gentleman closely engaged in the city, was 37 years of age, about 5 feet 7 inches in height, and 10 stones 2 lbs. in weight, of sallow complexion, but healthy aspect. He said that he had enjoyed excellent health until the age of 29, when he had a severe attack of fever, which left him in shattered health and extremely nervous. Some months elapsed before he regained his strength and was able to resume business, and he had suffered ever since, on slight occasions, from disturbance of the digestive organs, as evidenced by flatulence after meals, palpitation, colicky pain in the abdomen, and a tendency to acidity and diarrhœa. Accompanying these symptoms there was a distressing sense of depression, a disinclination to exertion, and an inability to apply himself steadily to work. He was always fearing some impending calamity, some serious and irremediable bodily affliction, or some loss or misadventure in business. His skin was cool and rather inactive; pulse 84. Tongue coated; bowels at one time costive, at another relaxed. The urine was frothy or firmly beaded, clear, acid, of pale sherry colour, sp. gr. 1028. It had a strong urinous smell, and was perfectly free from sugar and albumen. Nothing noteworthy was observed under the microscope; on the addition of nitric acid an intense effervescence ensued, and within half an hour crystals of nitrate of urea began to form and soon filled two thirds of that portion of the tube occupied by the urine. In this case, as in the last, Dr. Dickinson kindly determined the amount of urea excreted in twenty-four hours by examining a sample taken from the entire quantity of urine (1359 c.c.) which had been passed, and he found that it amounted to 55·1 grammes, or 22·1 grammes in excess of the average. In four other cases, in which, at my request, Dr. Dickinson determined the quantity excreted in twenty-four hours, it was found to amount to 42·5 grammes, 41·8 grammes, 39·06 grammes, and 51 grammes.

Now, all the instances which have come under my notice have had several features in common. With one exception,

that of a lady, aged forty-three, the patients have all been males, varying in age from twenty-three to fifty-four. Most of them have been essentially meat eaters, partaking sparingly of bread and other varieties of farinaceous food, but five of them ate meat only twice daily, and one, a member of our own profession, declared, not only that he never touched meat except at dinner, but that even then the principal part of his meal consisted of vegetable and farinaceous food. Without exception, the patients have been tolerably healthy in appearance, and often somewhat florid, though in some instances they have lost flesh slightly; indeed, their aspect has been so little indicative of disease, and their complaint of suffering has been so urgent, that any practitioner who did not examine the urine for urea would have surely regarded them as simply hypochondriacal. Usually the pulse has been quick and rather weak, and the patients' complaint has been of languor, flatulence, restlessness at night, and extreme nervousness. They have been afraid of encountering the rubs of everyday life, and in some instances have for the time lost their pleasure in society; they have eaten fairly, though with less than their usual relish, and the feeling of languor which has oppressed them, and of fatigue which has followed even a moderate amount of exertion, has led to their abstaining almost wholly from exercise. They have usually complained of extreme depression in the morning, with a sense of heat about the eyes, muzziness in the head, and weariness after breakfast—feelings which have in some measure passed off as the day has advanced, and they have partaken, as usual, of stimulants. The condition of the urine has varied; at one time it has been scanty, and loaded with lithates; at another abundant, or even excessive in quantity, of a light sherry colour and strong urinous odour; and these alternations have been repeatedly observed in the same case at intervals of a few days, or even of a few hours. The pale and clear urine, of a specific gravity varying from 1029 to 1032, and quantity from 32 oz. to 64 oz., is that which has usually contained the largest quantity of urea, and it has remained markedly frothy or beaded for twenty-

four or thirty-six hours, just like diabetic or highly albuminous urine. Dr. Prout has stated [1] that "urine containing a large proportion of urea is prone to decomposition, and generally soon becomes alkaline;" but in the cases under consideration the urine, even when exposed to the air, will remain acid and free from decomposition for two or three weeks in warm weather, and in cold weather will remain unchanged for as many months.

The method I have adopted in testing the urine has been to add to a small quantity of it in a test-tube about one third of its bulk of nitric acid, and then to shake the tube until all effervescence has subsided. The tube is then left at rest, to allow of crystallization of nitrate of urea to take place. In cold weather this is commonly effected in twenty minutes or half an hour, and it will usually occur in about the same time even in hot weather if the test-tube is immersed in a bath of cold water.

It should, perhaps, be added that the tendency to a recurrence of this form of disease is very great, and several of the patients have consulted me in reference to the same train of symptoms on more than one occasion.

The treatment adopted has varied in its details, but that which has proved most successful is based upon the view which I take of the pathology of the disease, namely, that it is connected primarily with perverted nervous action, in consequence of disturbed assimilation. Neurine tonics, such as arsenic, quinine, strychnia, iron and zinc, together with the mineral acids, the shower bath, and the dripping sheet, are the remedies which have proved most successful in my hands, and when combined with alterative doses of blue pill and colchicum will usually afford relief. In one or two instances, characterised by extreme irritability, the bromide of potassium, in conjunction with iron, has proved a more effective remedy, and occasionally the hypophosphite of soda has been serviceable.

The form of disease which I have thus endeavoured to bring before the Society has not as yet received distinct

[1] 'On the Stomach and Urinary Diseases,' 3rd edit., p. 94.

recognition, though the peculiarity of the symptoms by which it is signalised are such, I think, as entitle it to be regarded as a special form of derangement. Except in Dr. Prout's classical work on 'Stomach and Urinary Diseases,' I find no mention made of it in any treatise on the urine published in this country. Even Dr. Prout regards it as very rare, and, while suggesting its close connection with diabetes mellitus, declares (p. 96) that "where he has seen one case of an excess of urea in adults, he has seen twenty cases of diabetes." Dr. Parkes, referring to Dr. Prout's observations on these cases of azoturia, distinctly states,[1] "I have never seen a disease of this kind." It is obvious, therefore, not only that this condition of the urine has, in many instances, escaped recognition, but that the form of derangement on which it depends requires careful investigation.

The symptoms by which it is characterised appear to me to necessitate a complete revision of the theory hitherto accepted respecting the cause of the presence of urea in the urine. Up to the present time it has been held that urea is formed principally, if not solely, during the destructive processes of assimilation as a result of oxidation of the tissues, and in confirmation of this opinion most authors have asserted that it is found in largest quantities after violent exercise and during the excitement of febrile disorders, when the waste of tissue is at its maximum.[2] But I altogether doubt this statement, and certainly in the class of cases now brought before the Society this condition of affairs is reversed. The patients ordinarily remain in fair bodily condition, lose very little flesh, and bear the aspect of health; they are languid and lethargic while the attack is present, and take little exercise; their appetite is good, though their zest for food is impaired; and their principal complaint is of dyspepsia, prostration, and nervous derangement, so that they are often regarded as simply nervous and hypochondriacal. In short, it would appear that the azoturia must have some

[1] 'On the Urine,' p. 374.

[2] See Parkes, Beale, Hassall.

other source than the wear and tear of the tissues, and there are only two probable explanations of its occurrence—the one that it is referable, in part at least, to the production of urea from the elements of the food during the primary processes of assimilation, as a consequence of perverted nervous action; the other that a larger quantity than usual of the nitrogenous elements are secreted by the kidneys in consequence of a defective secretion of nitrogenous matters by the bowel. The recent experiments recorded by Dr. Parkes[1] would seem to show that this latter is at least a possible explanation, and unless, in any given case, the urine and the excreta from the bowels could both be examined, it cannot be affirmed positively that it does not afford a solution of the mystery. But it appears far more feasible to suppose that a certain portion of the food, instead of being utilised in the system, undergoes conversion into urea, and is then excreted without having fulfilled its mission in the animal economy. In the class of cases under consideration, the bowels are not more than ordinarily torpid, nor is there any evidence of defective intestinal secretion, whereas there are marked symptoms throughout of disturbed digestion and of perverted nervous action.

Several circumstances lead me to believe that this affection is more nearly allied to gout than it is to diabetes, as suggested by Dr. Prout. In the first place, not a trace of sugar was detected in the urine in any instance, though it was invariably sought for. Secondly, no less than eleven out of the twenty-seven patients were the offspring of gouty parents, and three of them had actually suffered from gout. Thirdly, the urine was often highly charged with lithates, and occasionally threw down crystals of uric acid, indicating a strong tendency to the lithic acid diathesis. Fourthly, in most instances the quantity of urea appeared to vary inversely as the quantity of uric acid. I speak hesitatingly on this point, because, unfortunately, the amount of uric acid in each specimen of urine was not determined by analysis; but when the urine proved, as it usually did, clear and often pale at

[1] See 'Med. Times and Gazette,' April 13th, 1867.

one time, and high coloured and loaded with lithates at another, the urea was invariably found to exist in largest quantities, as shown by the crystallization of nitrate of urea in those specimens of urine which were free from lithates, although the quantity of urine voided on those occasions was larger than when the lithates were deposited. If, on further examination, a direct relationship should prove to exist between the quantity of urea and uric acid excreted in these cases, another link would be added to the chain of events which appear to exercise a bearing on the causation of gout. At present my information on the subject is too imperfect to warrant such a conclusion, but I have deemed it expedient to draw attention to the facts as far as I have hitherto ascertained them.

In conclusion, I would express my regret that the difficulties which are encountered in private practice in inducing patients to preserve the whole of the excreta from the kidneys and bowels have prevented my ascertaining, by actual analysis, the cause of the excessive quantity of urea in the urine. Hospital practice, in which precise observations can alone be carried out with certainty and regularity, furnishes comparatively few of the class of cases under discussion, one only out of the twenty-seven cases having occurred amongst my patients at St. George's Hospital. But now that attention has been drawn to the subject, it probably will not be long before some gentleman has the opportunity of supplying what I feel to be a deficiency in this communication.

Appendix, *July 7th*, 1868.

Since the above paper was communicated to the Society, I have had the opportunity of verifying my observations in thirty-three additional instances, in four of which the subjects were females. These cases have not thrown any additional light on the nature of the disorder, but they have served to confirm the opinion I have propounded as to its origin, and also to show that this form of malady is essentially distinct

from that variety of azoturia which is accompanied by rapid wasting, of which Dr. Sydney Ringer's case affords a notable example.

I would also add that my attention has been drawn to the fact that Dr. Handfield Jones published some pertinent remarks on azoturia in a paper which appeared in the 'British Medical Journal' for October 12th, 1861, and that he afterwards enlarged upon them in his work on 'Functional Nerve Disorder,' pp. 438-441. Dr. Sieveking also discussed the subject in the 'British Medical Journal' for June 3rd, 1865, suggesting that the disorder described by Dr. Golding Bird as oxaluria was in truth azoturia, the malady now under consideration.

I.

ON CERTAIN POINTS

IN THE

ANATOMY AND PATHOLOGY

OF

BRIGHT'S DISEASE OF THE KIDNEY.

II.

ON THE INFLUENCE

OF THE

MINUTE BLOOD-VESSELS UPON THE CIRCULATION.

BY

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It is now generally admitted by pathologists and practical physicians that cases of *chronic Bright's disease* may be arranged in two distinct classes. The first class includes cases in which the kidneys after death are found of small size, of red colour, and with an uneven granular surface; while in the second class are included those cases in which the kidneys are large, white, and smooth on the surface. In a paper which was published in the 42nd vol. of the 'Medico-Chirurgical Transactions' I pointed out the chief features in the anatomical characters and in the clinical history of the two classes of cases by which they may be distinguished from each other.

Although there is now a very general agreement as to the doctrine that a small granular kidney is essentially different from the large, white, smooth kidney, yet there exists some diversity of opinion as to the minute anatomy and pathology of the kidney in the various morbid conditions of the organ which are included under the head of chronic Bright's disease, and I desire now to correct what I conceive to be the error of some doctrines which have recently been promulgated.

Some pathologists maintain that the small granular kidney is the result of an *intertubular deposit*. I believe this to be an error, and for the following reasons. Those who advocate this doctrine give no sufficient proof of an intertubular deposit, and they take little or no account of those remarkable intratubular changes which have been described and demonstrated by others.[1] In my own numerous examinations of small granular kidneys I have found no evidence of an intertubular deposit, but, on the contrary, in the wide spaces which commonly exist between the tubes I have seen only the atrophied remains of tubes and capillaries, with occasionally a doubtful appearance of thickening of the matrix. Perhaps the most conclusive proof of the absence of an intertubular deposit is to be found in the fact that these small granular kidneys retain their red colour and their vascularity even when the disease has reached a very advanced stage. This form of diseased kidney is commonly called the "*red* granular kidney," in contradistinction to the "large *white* kidney." Now, this red colour of the kidney is utterly inconsistent with the doctrine of an intertubular deposit. Any deposit between the tubes must of necessity obliterate the intertubular capillaries, and the seat of the deposit consequently become pale and exsanguine. In proof of this statement I need only refer to a form of disease in which the intertubular capillaries *are* filled by an intertubular deposit, namely, the so-called "fibrinous deposits" in the kidney. In the 9th volume of the 'Pathological Transactions' I have shown that these so-called

[1] See two papers, one by the author and one by Mr. Simon, in the 30th vol. of the 'Medico-Chirurgical Transactions.'

fibrinous deposits originate in the formation of coagula in the intertubular capillaries. In consequence of this obstruction there is an exudation from the overgorged Malpighian capillaries into the tubes, and the result is the formation of a firm, elevated, circumscribed patch, which is quite pale and anæmic, and which subsequently softens and becomes absorbed, leaving a cicatrix on the surface of the kidney.

The contrast between these anæmic patches, where an intertubular deposit can be proved to exist, and the red, vascular, cortical tissue of a small granular kidney, where an intertubular deposit has been supposed to occur, is very striking, and would of itself be sufficient to disprove the doctrine in question.

It has been said that the disintegration of the glandular epithelium of the convoluted tubes, which I have described as the primary and most constant phenomenon in the small granular kidney, is the result of post-mortem changes. This statement, however, is entirely disproved by the fact that "granular casts," composed of epithelium more or less disintegrated, are almost constantly present in the urine, and afford conclusive evidence that the disintegration of the gland-cells is in continual progress during the lifetime of the patient. The appearance of these granular casts, their number and their size, form important elements in our means of diagnosis and prognosis in this class of cases.

Another doctrine which I believe to be erroneous was first published by Virchow; I allude to the doctrine that the structural changes in the so-called waxy or amyloid degeneration of the kidney are the result of a primary degeneration of the minute arteries and the Malpighian capillaries. This brings me to the main object of this communication, which is to explain the remarkable changes which the minute blood-vessels undergo during the progress of chronic Bright's disease. My explanation of these changes will lead me to correct what I conceive to be the error of Virchow's doctrine.

In a paper which was published in the 33rd volume of the 'Medico-Chirurgical Transactions,' I first described a re-

markable condition of the minute blood-vessels of the kidney which I had observed in cases of chronic Bright's disease, namely, hypertrophy of the muscular coats of the minute arteries, and thickening of the walls of the Malpighian capillaries while the intertubular capillaries and veins present no appearance of thickening. Since the publication of that paper I have examined numerous specimens of diseased kidneys, and the result has been to confirm, in nearly every particular, the anatomical accuracy of the description there given.

In the paper referred to I stated that "the remarkable hypertrophy of the muscular coats of these vessels [namely, the arteries] seems to have for its object to assist in driving the blood onwards through the intertubular vessels in which the impediment exists."

I have long been aware that this explanation of the phenomena is physiologically untenable. It is now generally known and admitted that the minute arteries throughout the body, by the contraction of their muscular walls, have the effect of diminishing and retarding the blood-stream. The contraction of the small arteries, so far from assisting to propel the blood onwards, is, in fact, directly antagonistic to the heart's contractions, and tends to check the current of blood. The minute arteries by their contraction act as stop-cocks, while by their relaxation they permit a fuller and a more rapid stream of blood, but they have no direct propelling power. The heart is the force pump which drives the blood onwards indiscriminately through the larger arterial canals. The minute arteries, by their contractile power, regulate the blood supply in accordance with the requirements of each organ and tissue. This is the doctrine which is now generally received by physiologists.

Having recently been engaged in making a careful revision of the morbid anatomy and pathology of Bright's disease in its various forms and stages, I believe that I am now in a position to give the true physiological explanation of the hypertrophy of the muscular walls of the small renal arteries, which is so constant a phenomenon in the advanced stages of all the forms of chronic Bright's kidneys. My explanation

is this :—In proportion to the destruction of the renal gland-cells, and the consequent diminution of the secretory power of the kidney, there is less demand for blood to be acted upon by the gland; the small arteries consequently contract upon their contents, so as to maintain the balance between the blood supply and the diminished secretory action of the kidney. This continued over-action of the small arteries in antagonism to the heart results in hypertrophy of their muscular walls. I believe that all the facts will be found to harmonise with this theory.

That the thickening of the walls of the small renal arteries in these cases of chronic Bright's disease is a genuine instance of hypertrophy of muscular tissue is indisputable. The two layers of fibres—an inner longitudinal, and an outer circular—retain all their characteristic appearances, and are simply increased in thickness, while the arteries, being elongated as well as thickened, often become more or less tortuous. It is equally indisputable that hypertrophy of muscular tissue implies long-continued over-action of the tissue in question. Over-action of the muscular walls of the small arteries, of necessity, diminishes the supply of blood. And it seems probable that the diminished blood supply is the result of a beneficent compensatory action. For since a kidney whose secreting structure is partially spoiled by disease is incapable of acting upon the same amount of blood as a healthy gland, it is likely that an undiminished supply of blood to a diseased kidney would tend to embarrass the organ, through engorgement of its capillary vessels, while more or less of the blood would pass through the kidney without being freed from those peculiar constituents which it is the function of the gland to secrete.

The best and purest specimens of hypertropied renal arteries are usually to be found in the advanced stages of the small granular kidney. In the large white kidneys the arteries are almost constantly found more or less hypertrophied, but in the advanced stages of the disease, more especially when the kidney has the characters of the so-called waxy or amyloid degeneration, the walls of many of the

small arteries assume a white and wax-like appearance, and their muscular structure is thereby rendered confused and indistinct. But that this is a secondary and not a primary phenomenon is proved by the fact that, in the early stage of this waxy degeneration, while the gland is already much enlarged and presents the unmistakable features of the waxy kidney, the arteries are either unchanged or their walls are simply hypertrophied. The waxy appearance of the arteries and the Malpighian capillaries in the advanced stage of this form of Bright's disease appears to be a result of infiltration of albuminous or fibrinous material into the walls of the vessels.

In my former communication on this subject I spoke of the walls of the Malpighian capillaries as being *hypertrophied.* Now, although it is probable that the walls of capillaries which are long subjected to an increased pressure of blood may grow in thickness and in strength, it is nearly certain that the thickening of these Malpighian capillaries is mainly caused by the continued transudation of albumen and fibrin through their walls, and the degree of thickening appears to bear a relation to the amount of albumen in the urine during life. Virchow was the first to show that the blood-vessels in the white waxy kidneys are readily and deeply tinged by a solution of iodine. It has been abundantly proved that his application of the term *amyloid* to this form of degeneration was a chemical error.

I have said that, although the muscular walls of the arteries become hypertrophied in the advanced stages of all the forms of Bright's disease yet, that the purest specimens of hypertrophy are found in the small granular kidneys. This fact would alone render it nearly certain that the hypertrophy of the arterial walls is not a result of an impeded circulation through the intertubular capillaries, for we have evidence that there is a greater impediment to the circulation through the intertubular capillaries in the large white kidney than in the small granular kidney. The amount of albumen which transudes mainly through the walls of the overgorged Malpighian capillaries affords a good index of the degree of ob-

struction in the intertubular vessels. It is a well-known fact that the urine secreted by a large white kidney is, as a rule, much more highly charged with albumen than that secreted by the small granular kidney; indeed, the urine in the latter form of disease is often but scantily albuminous, and sometimes entirely free from albumen. It can scarcely be doubted that one main cause of the obstructed circulation through the intertubular capillaries of the large white kidney is that the greater number of the tubes, being enlarged by intratubular growth and exudation, compress and obstruct the capillaries which lie between them. This enlargement of the tubes does *not* occur in the small granular kidney, except in the case of the comparatively few tubes that become dilated by accumulated secretion and thus grow into cysts. It appears, then, that there is not a direct, but rather an inverse, relation between the hypertrophy of the arterial walls and the obstruction of the intertubular capillary circulation; we infer, therefore, that the force which calls for that over-action of the renal arteries, and which results in hypertrophy of their muscular walls, has its place, not in front of these small arteries, but behind them; in short, that the arteries become hypertrophied in consequence of the resistance which they continually offer to the propulsive force of the heart.

Another fact which is inconsistent with the supposition that the thickening of the arterial walls is a consequence of an impediment in the intertubular capillaries, is that the efferent vessels of the Malpighian bodies present no appearance of thickening or dilatation. It is manifest that any obstruction to the flow of blood through the intertubular vessels would react backwards first upon the efferent vessels, and with the same force as upon the afferent arteries.

The comparative scantiness of the albumen in the urine secreted by the small granular kidneys appears to be a result partly of the absence of great obstruction in the intertubular capillaries; partly it is due to the resistance which the well-nourished arterial walls offer to the heart's impulse.

And now, if we examine the state of the heart in these cases of chronic Bright's disease, we find that in the majority

of cases its muscular walls, more especially those of the left ventricle, are more or less hypertrophied, even though the valves of the heart and the walls of the large arteries be healthy, and, therefore, offer no impediment to the circulation.

The frequent coexistence of cardiac hypertrophy with chronic renal disease was first pointed out by Dr. Bright, in the first volume of the 'Guy's Hospital Reports,' and he suggested two explanations—"Either that the altered quality of the blood affords irregular and unwonted stimulus to the organ immediately, or that it so affects the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system."

We now find a very remarkable phenomenon, namely, hypertrophy of muscular tissue at the two opposite extremes of the arterial system, the propulsive power of the heart and the resisting force of the arteries—at any rate, that of the renal arteries—being simultaneously increased, and, without doubt, these two antagonising hypertrophies are mutually interdependent, the one being provocative of the other. It cannot be supposed that the great hypertrophy of the left ventricle, which is often found in cases of chronic Bright's disease, is a direct result solely of the resistance offered by the renal arteries. We must look for the cause of this hypertrophy rather in the fact that the blood, in consequence of degeneration of the kidney, being contaminated by urinary excreta and otherwise deteriorated, is impeded in its transit through the minute arteries throughout the body. We have evidence of such an impediment in the full, hard, throbbing pulse, which is a very common phenomenon in the advanced stage of chronic Bright's disease. Dr. Sanderson, in his recently published 'Handbook of the Sphygmograph' (p. 80), states that in cases of chronic Bright's disease with hypertrophy of the left ventricle, the sphygmograph affords decided evidence of increased arterial pressure. Still more conclusive evidence of an impediment to the circulation is afforded by the existence of hypertrophy of the muscular walls of the *arteries* in various tissues and organs. Dr. Hughlings

Jackson lately did me the favour to bring me a kidney, some vessels from the brain, and a portion of pia mater, from a man who had died with a contracted granular kidney. There had been headache and other cerebral symptoms during life, and after death we found that, not only were the walls of the renal arteries hypertrophied, but also, though in a less degree, those of the brain and pia mater.

In another case of chronic Bright's disease, with hypertrophy of the left ventricle, I found the muscular walls of the arteries of *the small intestines* much hypertrophied. And more recently, in another case of the same kind, after injecting the radial artery with a blue fluid, I found very marked hypertrophy in the walls of the small *subcutaneous arteries* over the back of the hand.

An impeded circulation through the subcutaneous arteries affords a probable explanation of the dry and inactive state of the skin which so commonly exists in cases of chronic Bright's disease, and of the resistance offered to the action of even so powerful a diaphoretic as the hot-air bath.

Diligent and careful observation will, without doubt, soon increase the anatomical evidence of arterial resistance to the circulation in cases of chronic Bright's disease. Action and reaction are equal, force must be met by an equivalent amount of force, and we shall probably find that the greater the hypertrophy of the left ventricle of the heart the greater and the more general is the hypertrophy of the arterial walls in various tissues and organs throughout the body.[1]

If I have correctly described and interpreted the structural changes in the minute arteries of the kidney, and if I have given a true account of the influence exerted by the muscular walls of the small arteries upon the circulation, then it can scarcely be doubted that the same physiological principle will be found applicable to a number of pathological phenomena. The sudden alternations in the colour of the face under the influence of mental emotion can scarcely be considered pathological; yet, in passing, it may be well to refer to the phenomena as a well-known and generally admitted

[1] See postscript, p. 76.

illustration of the fact that a rapid alternation of extreme anæmia and extreme turgescence of the capillaries may result from an alternate contraction and relaxation of the small muscular arteries.

Nearly a century ago Hales[1] proved, by experiments on animals recently killed, that while warm water passes very readily through the small vessels, cold water, decoction of bark, or a spirituous liquid, passes much more slowly. These experiments are explained by the principle that the small arteries, by their contraction, arrest or retard the passage of irritating or constringing liquids.

Blake[2] experimented on living animals by injecting various solutions into their blood-vessels, and he found that certain substances injected into the veins of a dog in a liquid form arrested the flow of blood through the lungs so rapidly that the animal died in less than a minute. Thus, for example, he injected fifteen grains of pure soda dissolved in six drachms of water into the jugular vein of a dog. In six seconds after the first portion of the injection was introduced the pressure in the systemic arteries began to diminish, and the mercury in the hæmadynamometer speedily sank to zero, although the pulsations of the heart still continued. In forty-five seconds the animal was dead. On opening the chest the left auricle and ventricle were found still contracting, but nearly empty, while the right cavities were so distended that their contraction was impossible; yet they too began to contract when a portion of the accumulated blood had been allowed to escape through a puncture of the heart. In another experiment he found that a hæmadynamometer in a *vein* indicated an increase of pressure at the same time that the arterial pressure was lessened. It is manifest, from these experiments, that paralysis of the heart was not the cause of the arrest of the circulation. In contrast with the effect of *soda* upon the circulation Blake found that *a solution of potash, or its salts,* or a solution of *sulphate of magnesia,* speedily arrested the heart's action. After death, thus

[1] Hales' 'Statical Essays,' 1769.

[2] 'Edinburgh Medical and Surgical Journal,' vols. liii, liv, and lvi.

produced, the left cavities of the heart were found filled with blood of a bright scarlet colour, affording, as he says, a certain proof that the movements of the heart had stopped before the respiration had ceased; while the only probable explanation of the effect of the soda injection is that the minute pulmonary arteries, by their contraction, arrested the passage of the contaminated blood.

In like manner, when air is forcibly blown into the vein of a living animal, or when in the human subject it is accidentally sucked in with the blood during the performance of an operation at the root of the neck or in the axilla, speedy death may result from the arrest of the frothy blood by the minute arteries of the lungs.

I have elsewhere endeavoured to prove that the arrest of blood in the minute arteries of the lungs *during the collapse stage of cholera* admits of but this one explanation; yet so entirely has the potent influence of the minute arteries upon the circulation been ignored by pathologists that, in their endeavour to explain the undoubted fact that the blood is impeded in its passage through the lungs of a cholera patient, they have asserted, in opposition to indisputable facts, either that the heart is paralysed or that the blood has become too thick to pass through the minute vessels.

Some of the phenomena of apnœa[1] admit of complete explanation only by reference to the contraction of the small arteries. Thus when, in consequence of imperfection or complete suspension of the respiratory changes, unaërated black blood circulates through the systemic arteries, these arteries become distended, there is a full and throbbing pulse, and the hæmadynamometer shows an increase of pressure upon the arterial walls. The increase of pressure in the systemic arteries continues, as Dr. Reid states, for some time after the animal has ceased to struggle, and when the attempts at respiration are few and imperfect. This distension of the arterial trunks must surely be a consequence of contraction of the minute systemic arteries excited by the circulation of black blood. But when the respiratory changes

[1] The term "apnœa" is here used advisedly instead of "asphyxia."

are entirely suspended or greatly interfered with the increased pressure in the systemic arteries does not long continue, for the circulation through the lungs becomes more and more impeded and soon entirely arrested. If, when the circulation has nearly ceased, air be readmitted to the lungs the blood again instantly moves on. Upon this point I extract the following from Dr. John Reid:[1]—"If a tube, with a stop-cock upon it, be tied into the trachea of a dog, and the stop-cock turned to exclude fresh air from the lungs until the circulation of dark blood along the arteries has become much enfeebled, *instantly*, on the admission of fresh air into the lungs, the blood springs from the cut artery of a bright red colour, and with greatly increased force." Now, I maintain that the only probable explanation of these phenomena is that when the respiratory changes are interfered with the minute branches of the pulmonary arteries contract upon their contents and retard the blood stream, just as we have seen reason to conclude that the renal arteries keep back the blood when the secretory power of the kidney is impaired. On the other hand, as soon as the air is again freely admitted to the lungs the minute pulmonary arteries, in obedience, probably, to an influence transmitted to them through the nervous system, instantly relax and the blood passes freely onwards. In giving this explanation of the phenomena of apnœa I agree with Dr. Reid in the opinion that the arrest of the pulmonary circulation depends primarily upon the suspension of the respiratory changes. I differ from him in maintaining that the main and immediate cause of the arrest of blood is the contraction of the small pulmonary arteries, which must surely be more powerful and more speedy in operation than any supposed attraction between the blood and the walls of the capillaries. Dr. Reid stated that the contractility of arteries manifests itself "by slow contractions, followed by equally slow relaxations." But this statement is certainly erroneous so far as relates to the small arteries. I have before referred to facts which indicate that both contraction and relaxation of the minute muscular arteries may occur

[1] 'Physiological, Anatomical, and Pathological Researches,' p. 46.

instantaneously. Mr. Lister[1] has proved by numerous experiments that the arteries in the web of the frog's foot may be made to contract and to dilate with extreme rapidity. Mr. Paget[2] has demonstrated the same phenomenon in the arteries of the bat's wing. On the other hand, we have reason to believe that any retarding influence which results from an altered relation between the blood and the walls of the capillaries comes on slowly, increases very gradually, and passes away again, if it pass away at all, as slowly and as gradually as it came on.

Chronic apnœa, as we all know, is invariably associated with engorgement of the pulmonary capillaries, and with more or less of watery exudation into the air-cells of the lung. In this state of chronic apnœa there is evidence of capillary stasis. On the other hand, in cases of acute and rapidly fatal apnœa the minute tissue of the lung is pale and comparatively anæmic. To illustrate this condition I performed the following experiment. A dog weighing 19¼ lbs. was killed by a ligature on his trachea; immediately after death the chest was opened. The right side of the heart was much distended, the auricle, in particular, standing out round and smooth, and having an elastic feel like that of an india-rubber ball distended with air. The left cavities were comparatively flaccid. A ligature having been placed round all the vessels, the heart was removed. On opening it 2 ounces of blood gushed from the distended right side, while 2½ drachms only escaped from the left cavities.

The lungs were pale, collapsed to an extreme degree, felt dry, and were crepitant; the right weighed 1¼ ounce, the left an ounce and half a drachm. The pulmonary artery, before the roots of the lungs were divided, was distended; the pulmonary veins were moderately full. The venæ cavæ were distended. After cutting across the great vessels 12 ounces of blood escaped into the cavity of the chest.

Here we have evidence of an arrest of blood in its passage

[1] "An Inquiry concerning the parts of the Nervous System which regulate the Contraction of the Arteries," 'Phil. Trans.,' 1848, p. 607.

[2] 'Surgical Pathology.'

through the lungs. It is evident that the arrest did not occur in the capillaries, for these vessels were nearly bloodless; the stoppage must have originated in the minute arteries, which by their contraction so opposed the passage of blood as to distend all the cavities and vessels leading up to the obstruction, and thus finally to arrest the circulation.

The extreme collapse of the lung when the chest is opened is a result of the extreme anæmia of its minute vessels, and the tough and non-crepitant feel of the lung is a result of the almost complete expulsion of the air from the vesicles during the extreme collapse after the chest is opened.

To illustrate the effect of post-mortem movement of the blood, another dog was killed in the same way by a ligature on the trachea, and the chest was opened after the dead body had been lying on its back for forty-four hours. The rigor mortis continued. The right cavities of the heart contained five drachms of blood; the left ventricle was firmly contracted, and contained a drachm of blood. The lungs were anæmic in their upper and anterior parts, dark and congested in the lower and back parts. Their weight was exactly the same as that of the first animal's lungs, but then it is to be observed that the first dog was a larger animal, his weight being 2 lbs. greater, and the capacity of his chest nearly twice that of the smaller dog.

In this second experiment it is possible that the distension of the right cavities of the heart had diminished after death, "the blood having made its way through various channels," as in Harvey's well-known observation on the criminal who had been executed by hanging.[1] And, further, some blood

[1] Harvey says, "I have several times opened the breast and pericardium of a man within two hours after his execution by hanging, and before the colour had totally left the face, and, in presence of many witnesses, have demonstrated the right auricle of the heart and the lungs distended with blood—the auricle, in particular, of the size of a large man's fist, and so full of blood that it looked as if it would burst. This great distension, however, had disappeared the next day, the body having stiffened and become cold, and the blood having made its way through various channels."—'Second Disquisition on the Circulation of the Blood,' Sydenham Society's Translation, p. 127.

had gravitated into the dependent parts of the lungs, which were thus rendered dark and congested.

This experiment illustrates the importance of opening the chest as soon as possible after death, when the object is to ascertain the distribution of blood in the heart's cavities and the state of the lungs at the moment of death. This precaution is especially important in the case of patients who have died in the collapse stage of cholera.

In the 'Lancet' of November 9th, 1867, Dr. Massey, of Nottingham, describes minutely the appearances found in the body of a man four hours after his execution by hanging. "On removing the sternum and cartilages of the ribs the lungs were not to be seen, but were found to occupy a very small space at the back part of the chest, resembling the contents of a fœtal thorax, the pericardial sac alone being seen. The colour of the lungs was of a darker hue than natural, especially at the bases. On cutting out the lungs a quantity of black fluid blood flowed. The structure was natural, but there was loss of crepitancy, and but very little air was contained in them. The right auricle of the heart was gorged to the greatest state of distension with blood, and the inferior cava was in the like condition; on opening the auricle a great quantity of black fluid blood gushed out. The right ventricle also contained a large amount of blood. The left auricle and ventricle were completely empty." It will be seen that the appearances described by Dr. Massey are essentially the same as those which I observed in the suffocated dog.

Anæmia of the minute tissue of the lung, with engorgement of the right cavities of the heart, will be found in all cases of rapid death from apnœa—for example, in some cases of rapidly fatal apoplexy or epilepsy, cases of spasmodic croup, and cases of sudden closure of the air-passages by a foreign body, or, more rarely, by rapid croupy or diphtheritic exudation.

The same physiological principle is applicable in all these cases. The circulation through the lungs is arrested in consequence of the suspension of the function of respiration, the arrest of the circulation being a direct result of arterial

contraction. That arterial contraction is the efficient cause of the arrest of blood is rendered still more probable by the results of some experiments to which I will briefly refer. Dr. Sanderson[1] poisoned a dog with woorara, the effect of which is to suspend the respiratory movements, while the heart's contractions are for a time unaffected. Death results from apnœa, but more slowly, Dr. Sanderson found, than when apnœa is produced by plugging the trachea, and the dynamometer in a systemic artery affords no evidence of increased pressure upon the arterial walls while black blood is circulating. Bernard has shown that this poison paralyses all the motor nerves, including those of the blood-vessels. The minute arteries, both pulmonary and systemic, having thus been deprived of their contractile power, offer no impediment to the passage of the blood; the circulation, therefore, continues until the heart's contractions gradually cease. In contrast with this experiment I may refer to one which was performed by Mr. Erichsen.[2] He pithed a dog in order to eliminate the disturbing element of violent struggling movements upon the arterial pressure. Then keeping up artificial respiration until he had ascertained the height of the mercury in the arterial dynamometer, he found that when the respiratory movements were suspended, and black blood consequently passed into the systemic arteries, the mercury rose to from two inches to two and a half inches higher than while respiration continued. The arterial pressure afterwards gradually diminished as the heart's action became enfeebled. In this experiment the resistance offered to the passage of black blood though the systemic arteries was clearly proved to exist after the respiratory movements had ceased and when the animal could not struggle.

The influence of the pulmonary vessels in arresting the circulation and so causing speedy death in apnœa receives indirect illustration from the curious fact, which was first discovered by Buffon, that the young of certain warm-blooded animals—for example, the dog, the cat, and the

[1] 'Proceedings of the Royal Society,' March 7th, 1867.

[2] 'Edinburgh Medical and Surgical Journal,' vol. lxiii, p. 15.

rabbit—may within a few days after their birth be immersed in water of moderate temperature for a period of sometimes half an hour before life is extinct.[1] The explanation of this interesting phenomenon is, without doubt, to be found in the fact that in the animals referred to the foramen ovale and the ductus arteriosus remain patulous for a few days after birth, so that when air is excluded from the lungs the blood passes directly from the right to the left side of the heart and to the aorta, and the circulation continues much longer than when, in older animals, the foramen ovale and the ductus arteriosus being closed, all the blood has to pass through the pulmonary vessels, and is thus subjected to their regulating and retarding influence.

And now a comparison of the phenomena of apnœa with those of renal disease appears to warrant the conclusion that an impeded circulation, the result of arterial contraction, may occur under two different conditions.

1st. When the respiratory changes in the lungs are impeded, or when the secretory action of a gland such as the kidney is impaired, the minute arteries of the lung or of the kidney—in obedience, probably, to a stimulus conveyed to them through the nerves from the capillaries—restrict and retard the blood stream. The anæmia of the pulmonary capillaries in cases of acute apnœa and the overgrowth of the muscular walls of the renal arteries in cases of chronic Bright's disease are results of one and the same physiological action of the arterial stop-cocks.

2nd. The minute arteries in any part of the body may be excited to contract by their contents becoming abnormal, and therefore more or less noxious to the tissues. We have an illustration of this in the resistance which the systemic arteries offer to the passage of unaerated black blood and of blood contaminated with urinary excreta. We have another illustration of it when certain foreign materials are either accidentally in the human subject or designedly in animals

[1] See Edwards, Dr. K., 'De l'Influence des Agens physiques sur la Vie,' 1824, p. 171; and Dr. J. Phillips Kay 'On Asphyxia,' 1834, p. 104, &c.

introduced into the veins and then arrest the flow of blood through the lungs.

We have yet another illustration of it, as I believe, in the effect of the cholera poison and its products upon the pulmonary circulation. It may be that even in this second class of cases, when contaminated blood is brought into contact with the inner surface of the arteries, the immediate incitement to the arterial contraction is a stimulus telegraphed to the arteries through the nerves from the capillaries of the tissues which are implicated.

The contraction of the minute arteries under the influence of a stimulus conveyed to them through the nerves is analogous to the contraction of the pupil which results from the stimulus of light upon the retina reflected through the motor nerves to the iris. It is also analogous to the forcible coordinate action of the respiratory muscles when there is an increased demand for air to combine with the circulating blood, or when a forcible expiratory effort is required to expel a foreign body from the air-passages.

The arrest of morbid blood in its passage through the vessels is probably the result of a law or principle which, in general, is as wholesome as the arrest of a foreign body in its passage through the larynx, yet in particular cases the arterial or the laryngeal spasm by which this generally wholesome arrest is effected or attempted may be itself the immediate cause of death.

The late Dr. Alison, and many pathologists since Dr. Alison, have taught that there exists in the minute blood-vessels a power by which, in various morbid states of the tissues and of the blood, the heart's action is antagonised, and the circulation of the blood is impeded; but it is remarkable that this retarding influence has been almost universally believed to exist in the capillaries which have no contractile power, while the stop-cock action of the small arteries, with their contractile, muscular walls, which physiologists now systematically teach, has been almost entirely overlooked and ignored by pathologists. One of the main objects of this communication is to direct attention to this

action of the small arteries in various pathological states of the system, and to afford anatomical as well as physiological evidence of its reality and its powerful influence. Henceforth, when we are investigating the subject of capillary engorgement and stasis, we shall have to inquire whether even this is not, in part at least, attributable to the diminished *vis à tergo* which must result from contraction of the minute arteries. To attribute capillary obstruction and stasis to some mysterious attraction or want of attraction between the blood and the minute vessels, while the action of the arterial sphincters is disregarded, is an inconsistency which would have its counterpart in a fanciful attempt to explain the retention of urine in the bladder by a supposed attraction or want of attraction between the urethra and the urine, while the action of the *sphincter vesicæ* is taken for nought.

As exceptions to the general disregard by pathologists of the influence of the small arteries upon the circulation, it is right to mention that Mr. Erichsen, in his very able and interesting article on asphyxia, published in 1845 ('Edinburgh Medical and Surgical Journal,' vol. lxiii), attributes the impeded transit of unaërated black blood through the systemic vessels to the contraction of the small arteries upon their abnormal contents. And Kussmaul and Tenner, in a monograph translated by the New Sydenham Society, having proved that anæmia of the brain is a common cause of epileptiform convulsions, refer to a number of facts which render it highly probable that in many cases of epilepsy the anæmia of the brain, which is the immediate cause of the convulsive paroxysm, is a result of a sudden contraction of the muscular walls of the minute cerebral arteries. This theory of arterial contraction as the immediate cause of cerebral anæmia in some cases of epilepsy has been adopted by Dr. Brown-Séquard, by Dr. Russell Reynolds, and by other pathologists. Lastly, the sphygmographic observations of Marey and his followers are based upon the principle that, while the blood is driven onwards by the heart, the minute arteries, by the contraction of their muscular walls, antagonise the heart and regulate the blood supply to the various tissues and organs.

POSTSCRIPT.—Since this paper was read we have found at King's College Hospital that in every fatal case of chronic Bright's disease with hypertrophy of the left ventricle there has been decided hypertrophy of the arterial walls in most of the tissues examined, not only in the kidneys, but also in the skin, the intestines, the muscles, and the pia mater. About ten cases have been examined. In some instances the kidneys have been small and granular, in others large, smooth, and pale; the hypertrophy of the arterial walls in the various tissues has been found in both classes of cases.

The facts hitherto observed all point to the conclusion that hypertrophy of the walls of the small arteries is a result of their continued overaction in opposition to the heart. Mere distension of the arteries in consequence of an obstruction in front does not cause hypertrophy of their walls. This is proved by specimens showing no thickening of the minute pulmonary arteries in emphysematous lungs, where there has been extensive obliteration of pulmonary capillaries, with hypertrophy of the right ventricle; and by other specimens showing no thickening of the arteries in the lung of a patient who had chronic incompetence of both the mitral and aortic valves, with much pulmonary engorgement and hæmoptysis. The arteries are best seen after injection with Prussian blue.

Dr. Kelly, the able pathologist of the hospital, has bestowed much time and labour upon the details of this interesting subject, and he will shortly publish the results.

I am indebted to my friend Dr. Hyde Salter for the accurate drawings of the arteries, both normal and hypertrophied, with which this paper is illustrated.

DESCRIPTION OF THE PLATES.

The arteries in all the specimens have their canals filled with Prussian blue. The magnifying power is 200 diameters in each case, except Plate II, Fig. 1, which is drawn from a specimen magnified 100 diameters.

PLATE I.

Fig. 1.—Normal artery from the pia mater.

Fig. 2.—Arteries with hypertrophied walls from the pia mater.

Fig. 3.—Artery with hypertrophied walls from the small intestines.

PLATE II.

Fig. 1.—Normal artery from the skin.

Fig. 2.—Artery with hypertrophied walls from the skin. It will be seen that the walls of the comparatively large artery (Plate II, fig. 1) have the same structure and about the same thickness as the walls of the smallest branches of the artery in this figure.

Fig. 3.—Artery with hypertrophied walls from muscle.

Fig. 4.—Artery with hypertrophied walls from the kidney. In the hypertrophied renal arteries there is always an inner longitudinal layer of fibres about equal in thickness to the outer circular layer. This thickened longitudinal layer has not been seen in the hypertrophied arteries from any other tissue.

N.B.—All the hypertrophied arteries are from the subjects of chronic Bright's disease.

Fig 1.

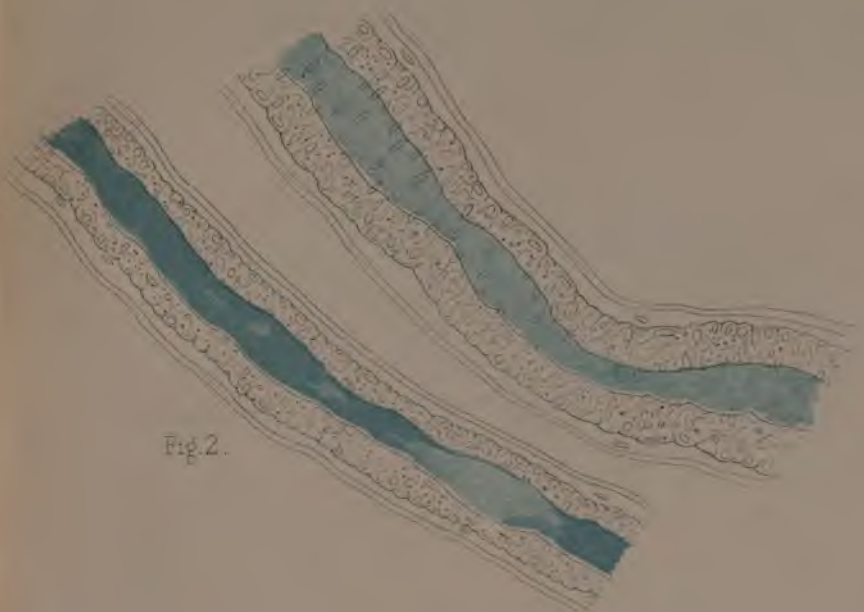

Fig. 2.

Fig. 3.

C.H. Ford. W. West. imp.

Fig 1.

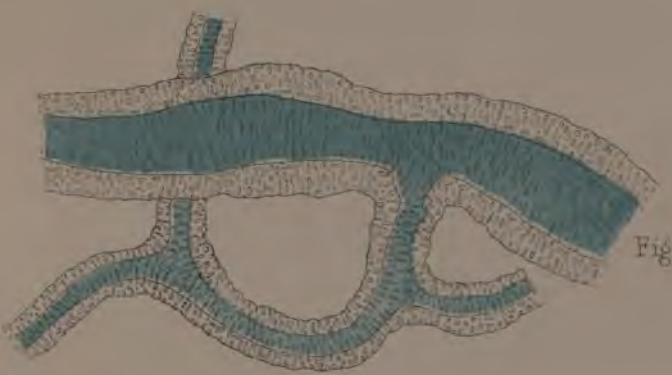

Fig 2

Fig 3

Fig 4

G.H.Ford

ON

THE CURE OF CLEFT PALATE

BY

OPERATION IN CHILDREN,

WITH A

DESCRIPTION OF AN INSTRUMENT FOR FACILITATING THE OPERATION.

BY

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Received Oct. 4th, 1867—Read Jan. 14th, 1868.

THE operation for the cure of fissured soft palate has been brought nearly to perfection by the labours of Sir William Fergusson. He first recommended and practised, as essential to success in the operation, the systematic division of those muscles that tend to draw asunder the margins of the cleft.

Professor Langenbeck, Mr. Pollock, Mr. Avery, Dr. Billroth and others, have done almost as much for the treatment of fissures of the bony palate, and thus it is that there is as little room as there is need of improvement in the modus operandi usually adopted for the cure of these deformities.

The object of this communication is, if possible, to extend the benefits of the operation (both on the hard and soft palate) to patients of tender years, to those deficient in physical courage or in the power of enduring bodily pain.

As the matter at present stands we have a good and successful operation, but very painful to the patient, and therefore limited in its application to individuals of adult age and of courageous and resolute disposition.

Referring to this subject Sir W. Fergusson writes,[1] "Unless the patient is remarkably steady and determined to permit the operation to be finished, it is useless to attempt it, and such fortitude is not to be expected before the age of puberty."

And, again, the same author in his lectures before the College of Surgeons, thus expresses himself:—"I have never attempted the operation in infancy, and consider the circumstances most favorable at or above puberty, but I have frequently operated between the ages of ten and fifteen[2] when patients have been steady and courageous. I have never operated under chloroform, and, whilst I do not deny the possibility of doing so, I am of opinion that, as a rule, it is absolutely necessary to have the patient conscious, so that he may facilitate the steps of the operation in various ways."

Mr. Pollock, in writing on the same subject in 'Holmes's System of Surgery,' thus expresses himself:—"The age of the patient is a matter of some consideration to the surgeon who proposes to operate for the permanent closure of a cleft palate. There can be no doubt, as a general rule, that the patient should be old enough to desire an operation, should have sufficient determination to endure it, and should be able to estimate the advantages derived from it. For many reasons the age of fifteen appears to be the earliest period for the operation; often a later period is desirable, though in some few instances it has been performed on younger children."

[1] 'Practical Surgery,' p. 530.

[2] 'Lancet,' 1864, vol. i, p. 723.

Mr. Pollock goes on to notice the tedious nature of the operation—the inadmissibility of chloroform—and recommends that, in the early years of life and up to the period of puberty, an artificial palate should be worn; the patient being subsequently allowed the refusal of an operation.

Langenbeck's [1] published opinion on this subject is to the following effect:—The earlier experiences of the success of staphyloraphy lead uniformly to the result that, before the expiration of the sixteenth year of life, the operation cannot be undertaken with any certainty of success.

Our experience shows that the operation can be employed at a far earlier age.

He goes on to say—"I fear, however, that for staphyloraphy the age of seven years will be a limit not to be overstepped as a rule, and that one must abstain from employing the operation in younger children."

Langenbeck states that simple staphyloraphy, *i. e.* the operation on the soft palate alone, has been recorded in eleven instances in children between the fourth month and sixth year of life, and in every case unsuccessfully;[2] while Billroth has succeeded in curing a cleft of both the hard and soft palate in a young infant in three operations, the first operation being undertaken when the child was but twenty-eight weeks old.

Of the performance of the operation says the same author:—"The difficulties of staphyloraphy in young children, though inexpressibly great, yet may be surmounted; the union of the wound in its whole extent can also take place, as I have in two cases experienced, though the nutrition of the child always suffers somewhat on account of the pain in swallowing not allowing them to take sufficient nourishment; this, especially, causes the separation of the sutures, and is, as appears to me, an insuperable difficulty. On this account children begin to fail about the sixth or eighth day after the operation; before the sutures are entirely separated, the suture holes begin to suppurate, and the soft palate that had united again separates."

[1] 'Lang. Archives,' Band. v, p. 43. [2] Loc. cit., p. 44.

The advantages of being able to cure cleft palate in childhood will at once be conceded by those who are familiar with the deformity, and the inconveniences it entails. The imperfections of deglutition, taste and smell, and the unnatural thirst, will probably be remedied at whatever period of life the cure of the deformity be effected. But it often happens that the union of the palate by operation is accomplished too late to do more than slightly improve the defective articulation, though it may cure the nasal resonance of the voice.

A resolute and intelligent adult patient, after a successful operation, may acquire by his own efforts, or with the aid of an instructor, the power of distinct and normal articulation. But in some the difficulty of unlearning an almost life-long habit is well nigh insuperable.

It is probable that were the deformity of cleft palate cured in childhood, before or whilst the power of speech is being acquired, there would be but little difficulty in teaching a child to articulate distinctly.

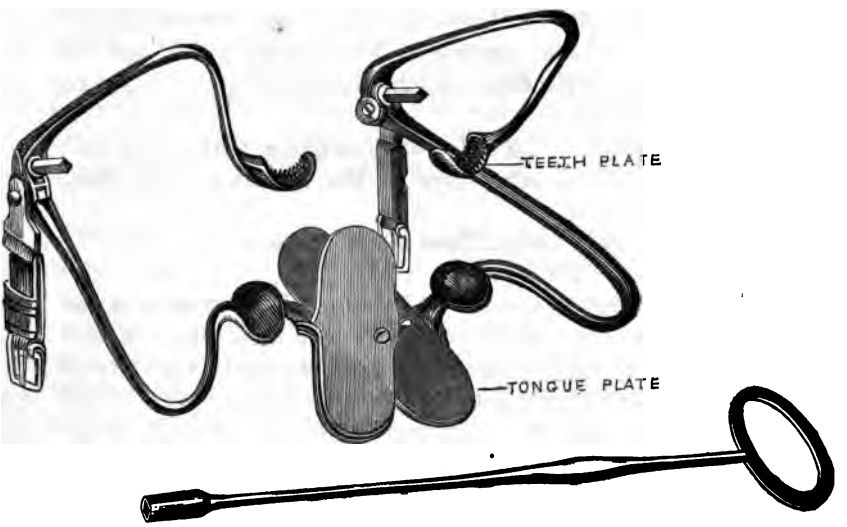
It is at least certain that the earlier the cure was effected, the less difficulty would the patient have in learning to speak plainly.

It is needless to occupy the time of the society in discussing the advantages of an early cure of the deformity, they will be conceded by all. I will at once, therefore, describe the plan by which I hope that these advantages may be obtained.

The plan which I venture to introduce to the society depends largely for its success on the employment of an instrument whereby the jaws are opened and fixed, the orifice of the mouth is dilated, and the tongue is held down; and this without in any way incommoding the operator, or encroaching on the small space at his command.

By the use of this instrument the patient is rendered perfectly helpless; the palate and fauces are brought plainly into view, and much more room is obtained for the manipulation of instruments than can be gained by the most strenuous voluntary efforts of the patient. Chloroform can and should be used, and thus the operation may be performed with far

greater rapidity and precision than when the surgeon is dependent upon the self-control of even the most courageous patient. The instrument in question, which is a species of gag, consists of a tongue-plate for depressing that organ; attached to this plate are two lateral expanding arms of a peculiar shape, which can be opened by a key working on a cog-wheel: these are for dilating the orifice of the mouth; they have teeth plates which act upon the jaws and open them. A strap is made to pass round the back of the neck, to keep the instrument in position.



This gag will be found useful in excision of the tonsils of unruly children, in operations on the pharynx and interior of the mouth. It can be used in operations on the tongue, but if employed for this purpose, it must be turned upside down, *i. e.* the tongue-piece must rest against the roof of the mouth.

I should think it might prove of service in laryngoscopy, and especially in operations on the interior of the larynx when it is important to economise space and secure a steady position.

I may be excused for shortly stating the details and order of operative proceeding that I have found most useful when performing staphyloraphy under chloroform.

It is important that the stomach should be quite empty, and equally so that the patient should be in no degree in want of food, so as to avoid both chloroform sickness on the one hand, and the necessity of taking food soon after the operation on the other.

The patient being under chloroform, in the recumbent position, in front of a window, his head may be supported on a pillow by an assistant standing behind the table. The gag being introduced, closed, and the strap drawn tight, the jaws and lips can be opened to the desired extent, and the instrument may be given into the hands of the assistant who holds the head.

The following is the order of proceeding that seems best adapted to the circumstances of the operation when done under chloroform.

1st. To pare the edges from below upwards.

2nd. To pass the sutures.

3rd. To draw up the palate holding all the sutures in one hand, and with the palate on the stretch to divide, if necessary, the palato-pharyngeus, and subsequently the levator palati; if the palate will not come easily together, two lateral oblique cuts may be made, one on either side above the highest suture, separating the soft from the margin of the hard palate to a small extent.

4th. To sponge out the parts, and tie the sutures beginning at the lowest.

5th. To avoid using the sponge, if possible, until the close of the operation.

Sickness occurring during the operation is an embarrassing circumstance, though by no means an insuperable difficulty. On two occasions I have postponed the operation on observing the child vomit while taking chloroform. On four occasions I have satisfactorily completed the operation notwithstanding the sickness.

The paring the edges of the fissure from below is impor-

tant to avoid being embarrassed by the flow of blood from above. It is well to postpone the division of the muscles until late in the operation for the same reason. By drawing the palate forwards by means of the sutures the muscles which prevent the approximation of the fissure are at once appreciated, and by thus putting them on the stretch their division is facilitated.

The use of the sponge to the palate, as is well known, increases the amount of tonsillar secretion, and is apt to excite vomiting. If the steps of the operation be done in the order above described, its use is seldom required until the last stage.

The material that I have found best adapted for sutures for the soft palate in the young, is *fine fishing gut*, if there be any tension on the suture; while *horsehair* has answered best for the lower part of the velum and uvula, or in any case where the sides of the cleft come easily together. Both these substances, if soaked for a few minutes in water, will become sufficiently supple for use. They have the advantage of being quite as unirritating to the tissues as silver wire; while being softer they cause less pain during the movements of deglutition; they do not abrade the tongue, and, unlike wire, they can be removed without giving pain to the patient, or disturbing in any way the newly united soft parts.

In fastening both gut and horsehair sutures, a third knot should be added to the ordinary double knot usually tied; this is a necessary precaution to be secure against slipping.

I have tried, with good result, the clamping down the suture with a perforated shot in lieu of tying: this is apt to cut the suture, unless a small glass bead be threaded on the sutures and passed down in front of the shot.

In regard to the unirritating nature of both gut and horsehair when introduced into the tissues, it may be worth mentioning, that in a little girl, aged nine, I removed the sutures more than a month after they were introduced; and at that time the wound was perfectly and soundly cicatrised, the sutures appearing to cause no sort of inconvenience or irritation.

With a view to simplify and expedite the performance of the operation I have found it convenient to dispense with the silk threads generally used for drawing the permanent sutures into position, and, instead, to pass the gut or horse-hair at once through both sides of the cleft palate.

This can be done by employing a fine and rather sharply curved tubular needle, or by using a needle in a handle bent at nearly a right angle, with an eye near the point. One can imagine that the width of the cleft might be so considerable as to prevent the passing of the sutures in the manner described, but when practicable, much time and trouble may be saved by the adoption of the plan.

In order to adapt the needles to the small dimensions and delicate structure of the soft palate in children, I have had them flattened laterally, instead of on the concave and convex aspect, as is usual in curved needles. This allows of the needle being made much finer without any loss of strength at the curve.

It is advisable in operating on children that forceps should be sparingly used to the margins of the palate. They were, therefore, only employed in passing the first or lowest suture. When this had been adjusted, it was used to draw the palate forwards, so as to bring the free edges somewhat together and within easy reach of the needle for passing the next suture. Thus, in turn, each suture was used to assist in adjusting the one next above it: this plan saves much trouble and avoids bruising the margins of the cleft.

In every case the principles recommended by Sir W. Fergusson have been carried out, though the plan used for dividing the muscles has been that practised by Mr. Pollock. That is, the levator palati has either been divided (or at all events its division has been attempted) through a puncture in front of the soft palate, the palato-pharyngeus has usually been notched and occasionally the palato-glossus. Where the soft parts were very scanty, and the tension considerable, oblique lateral cuts have been made at the upper angle of the fissure, separating the soft palate from its attachment to the bones for a small extent.

The patients operated upon have been allowed a liberal supply of wine and fluid nourishment after the operation, and have not been confined to their beds.

Appendix.

Appended is a table of eleven cases of operation on cleft palate, in all of which the gag was employed and chloroform was administered.

In all of these the operation of staphyloraphy was performed, that is, no attempt was made in any case to separate the muco-periosteum from the roof of the mouth, nor to unite the fissure of the hard palate.

Two of the cases only were simple uncomplicated fissures of the soft palate; the result in both of these was entirely successful.

The remaining cases were all complicated with fissure of the hard palate to a greater or less degree; of these, six were successful, and three unsuccessful.

The operations in the cases of uncomplicated cleft of the soft palate were, as one would suppose, easily and quickly performed. One of these patients was thirteen years and the other two years and eleven months old. The articulation of the younger child began to improve from the date of the operation. Of the elder child Dr. Priest reports, nine months after the operation, that she "continues to improve, her articulation is much better, and he hopes in a few months that very little trace of the infirmity will remain."

The subjects of complicated cleft of the palate—six in number—who were successfully submitted to operation were three, five, six, nine, ten, and twenty-seven years of age respectively. The operation in every case was performed upon the plan of which a description is given in the paper.

Inquiries have been made, whether there is not danger of blood getting into the larynx during the performance of the operation under chloroform? I can only reply that no symptoms have been observed during or after the operation

that would indicate that blood had entered the larynx, though frequently blood has been vomited after the patient has been put to bed. Indeed, there is but little bloodshed until the very last step in the operation, if the order of proceeding be followed that is here recommended.

I have counted as successful all cases where I have succeeded in curing the fissure in the soft palate. One of the cases recorded in this category still requires a small operation to unite the uvula, and in another there remains a small central hole in the soft palate; defects which can be remedied at the first convenient opportunity.

In no case was the union of the hard palate attempted by operation, and I believe that ere long I shall be in a position to prove that, provided the union of the soft palate be effected in early life, the operation on the hard palate will in many cases be unnecessary, and in some unadvisable.

The measurements that I possess showing the gradual contraction of the hole left in the roof of the mouth warrant me in anticipating thus much; though none of my cases are of sufficient duration to enable me thoroughly to realise my anticipations.

Thus in one case, No. 2, the operation was performed ten months ago, and the gap left in the hard palate measured exactly an inch in its long diameter ten days after the operation; seven months afterwards this hole measured $\frac{10}{16}$ of an inch.

In case 3, the operation on the soft palate was performed fourteen months ago, leaving a considerable hole in the roof of the mouth; this hole is now about the size of No. 6 catheter.

In case 7 the soft palate was united ten weeks ago, leaving a hole in the roof of the mouth measuring one inch in its long diameter; a month later this opening had diminished by a quarter of an inch.

I have, for the present, abstained from making any remarks on the effect of an early operation on the power of articulation, since my experience is much too recent to admit

of any well-grounded conclusions being formed on the subject.

Three instances of failure are recorded in the table of cases, one was in a child aged two years, who proved quite unmanageable; after the operation this infant did not cease to endeavour to tear out the sutures with his fingers.

Another failure was in the case of a girl, aged thirteen years, in whom I am at a loss to assign a cause of the non-union, unless it were that the levatores palati muscles were not satisfactorily divided.

The third, in whom the operation failed, was a boy three years of age, in whose case the separation of the wound was due to vomiting which came on the third day after the operation.

On a future occasion I shall hope to include all these as successful results.

An instance of successful operation on the palate of a woman, aged twenty-seven, has been tabulated, since I cannot but believe that the plan recommended is suitable to all ages; and I would venture to hope that in future chloroform will generally be administered for the performance of this operation.

Additional experience, for which I trust we shall not need to wait long, is required to determine the most favorable period of childhood at which the cure of cleft palate can be undertaken.

Case.	Name, age, sex, &c.	Nature of the deformity and state of the patient.	Operation.	Result.	Remarks.
1	— Cater (F.), æt. 13; St. Bartholomew's, March 27th, 1867.	Complete cleft of the soft palate. Speaks very indistinctly.	Under chloroform. Four gut sutures employed. Lev. palati and pal. pharyngei divided.	Complete union. Perfect symmetry of the palate.	Nine months afterwards articulation reported to be much better and improving.
2	L. Brown (F.), æt. 9; St. Bartholomew's, Feb. 22nd, 1867.	Cleft of all soft and half the hard palate. Speech unintelligible. Food returns through nostrils.	Chloroform. Soft palate closed with five gut sutures. Muscles divided and oblique cuts made on either side to relieve tension.	Wound united throughout. A hole one inch long between soft and hard palate. Seven months later this measured $\frac{10}{16}$ths of an inch.	Articulation much improved, but very imperfect. Food no longer returns.
3	C. Wilkins (F.), æt. 6; Children's Hospital, Nov. 4th, 1866.	Cleft of soft and of posterior third of the hard palate. Speech unintelligible. Food frequently returned by nostrils.	Gag used, without chloroform. Soft palate closed with four silver sutures. Muscles divided. Five months later three horsehairs were passed through lower part of palate.	Soft palate united, except lower part. Second operation quite successful. A small hole in the roof of the mouth.	Speech very much more distinct. Food does not return. Hole in the roof of the mouth the size of a No. 5 catheter.
4	J. Bartlett (M.), æt. 2; Children's Hospital, March 26th, 1867.	Complete cleft of hard and soft palate and alveolar process, with harelip. Cannot speak.	Chloroform. Soft palate closed with five gut sutures. Muscles divided. Two months later operation repeated.	Boy proved unmanageable. Nothing but uvula united. Unsuccessful.	This patient could not be prevented from dragging with his fingers at the sutures.

5	S. Brown (F.), æt. 2 yrs. 11 m.; Children's Hospital, August 27th, 1867.	Complete cleft of soft palate. Can only speak one or two words. Food rarely returns through nostrils.	Chloroform. Fissure closed with two horsehair and two gut sutures. Second operation, gut and horsehair used. Muscles divided.	Attacked with scarlatina the day after the operation. Unsuccessful. Successful.	Five months after the operation this child had learned to speak well, so the mother reports.
6	L. Aspley (F.), æt. 3 yrs. 2 m.; Children's Hospital, June, 1867.	Cleft of soft palate and posterior part of hard. Articulation very indistinct.	Chloroform. Soft palate closed by two gut sutures and one silver wire. Four months later the operation was repeated. A month later two sutures passed.	Scarlatina and hooping-cough succeeded the operation, and the result was unsuccessful. Half the palate united. Attacked by mumps. The soft palate is nearly completely united.	The power of articulation has much improved.
7	F. Cunningham (M.), æt. 10½; Children's Hospital, August 30th, 1867.	Cleft of soft palate and posterior part of hard. Articulates very imperfectly. Cannot be understood.	Chloroform. Soft palate closed by two gut and one horsehair suture. Muscles divided.	Soft palate united. A hole one inch in length remains in the roof of the mouth.	A month after the operation the hole in the roof of the mouth was three quarters of an inch in length.
8	H. Miles (M.), æt. 5; Children's Hospital, August 20th, 1867.	Double harelip and complete cleft of the hard and soft palate, with erection of columna nasi. Four years ago the harelip was cured, and the cleft in the hard palate has since greatly improved.	Chloroform. Soft palate united by one silver and four gut sutures. Muscles divided.	Union of all the palate except the lowest part and the uvula, which during the process of cicatrization came together.	Articulation greatly improved. The hole between the hard and soft palate is diminishing.

Case.	Name, age, sex, &c.	Nature of the deformity and state of the patient.	Operation.	Result.	Remarks.
9	M. A. Blake (F.), æt. 13; Children's Hospital, Oct. 28th, 1867.	Harelip and cleft of hard and soft palate. Harelip cured twelve years ago. Speaks very badly. Food occasionally returns.	Chloroform. Soft palate united by four gut and one horsehair suture. Muscles were imperfectly divided. Second operation similar to the first, four months later.	Three days after the operation the sutures had all ulcerated out. Unsuccessful.	The non-union in this case seemed due to some constitutional peculiarity, as the local conditions were unusually favorable.
10	A. Harding (F.), æt. 27; St. Bartholomew's, Oct. 30th, 1867.	A very wide cleft of the soft and half the hard palate. Articulation very imperfect.	Chloroform. Soft palate united by four gut sutures. Muscles divided.	Soft palate united, leaving a hole in the roof of the mouth 1⅛ inch long.	Three months later the hole in the palate had diminished to ⅝ths inch in length. Articulation had much improved.
11	E. Knight (F.), æt. 3; Children's Hospital, Dec. 16th, 1867.	Double harelip. Cleft of hard and soft palate. Two years ago the harelip was cured. Speaks very badly.	Chloroform. Soft palate united by four sutures. Muscles divided incompletely.	Vomiting came on two days after the operation, and the sutures all gave way.	Non-union in this case seemed to be due to the vomiting, though the incomplete division of the muscles was unfavorable to success.

CASE

OF

DOUBLE HERNIA IN A SINGLE SAC.

BY

HENRY LEE, F.R.C.S.,

SURGEON TO ST. GEORGE'S HOSPITAL.

Received Nov. 11th, 1867.—Read Feb. 11th, 1868.

J. S—, æt. 32, was admitted into St. George's Hospital under my care on the 7th of November, 1867. He had suffered from a rupture on the right side for ten years, and had worn a truss.

At six o'clock in the morning, previous to his admission, he fell on some timber, and immediately afterwards felt pain in his rupture and in the abdomen. He was brought to the hospital at 7.30. The pain then continued unabated and he vomited. The taxis was applied, and the tumour said to have been reduced in size.

At 1 p.m. a consultation was held upon the case. The swelling in the right groin was seen to consist of two portions; one of these, which in the following description will be called the *first*, had descended into the upper part of the scrotum, and was below the level of Poupart's ligament. The other, or *second* portion, was firmer to the touch, more painful, and situated above the level of Poupart's ligament. These two portions, which were very nearly in a vertical line, were separated by a depression distinctly visible on the surface of

the skin. The first swelling had a very free and distinct impulse communicated to it when the patient coughed, and could be handled without giving the patient much inconvenience. The second swelling received no impulse on coughing, and when handled gave him a sensation as if his testicle were being compressed. The patient in fact believed the second swelling, which had existed only eighteen months, to be the testicle, which, on the right side, had not descended into the scrotum. The second portion of the tumour, he said, altered its position from time to time. When the first swelling was down it was higher; when that was replaced, it was lower. He described very distinctly the fact that the first swelling would become reduced with a gurgling noise, and then that the second swelling would occupy its place. When the first swelling came down again, the second swelling would be forced again upward. But the two always remained separate and distinct.

The patient now (1 p.m.) complained of considerable pain in the abdomen and in the rupture. The taxis was again applied, but neither portion of the swelling could be reduced. The countenance was anxious, and the pulse accelerated. The bowels had not been moved for two days. As the impulse upon coughing remained so very distinct, it was considered that the bowel could not be strangulated, and that an operation would not be necessary. A turpentine injection was administered, and some ice applied over the tumour. At 2.30 the bowels had acted twice, and the patient expressed himself as feeling more comfortable.

Late in the evening the symptoms recurred with increased severity. The patient was repeatedly sick, the abdomen was tense and painful, and the rupture was more distended, and painful to the touch. The patient was restless, irritable, and anxious; pulse 95.

At 11.45 chloroform was administered, and the following operation performed: The skin and subjacent parts were divided over the first swelling. A rather tight constriction was found at the external ring; this was divided, but the hernia was as tense as before. The sac, which was very

thick, was then opened, and a considerable quantity of fluid escaped. The bowel which presented itself was congested and distended. It was traced up to the internal ring, which appeared to be at an unusual depth. The stricture was here felt by the point of the index finger, and divided with the hernia knife. The bowel now became flaccid, and its contents easily passed within the abdomen. The bowel itself was then reduced without difficulty.

The second swelling was now examined. It was found to be, not the testicle, as had previously been thought, but a distinct and rather large fold of intestine more distended and more congested than the first. These two portions of intestine were contained in the same sac. Upon tracing the second portion upwards, it was found to follow the same direction as the first, but to communicate with the abdomen by a very narrow separate opening. This was situated to the inner side of the opening through which the first portion of the intestine had passed, and had no connection with it. The second stricture was divided, and the second portion of bowel replaced without difficulty.

The inguinal canal was now felt to be distended above, where it terminated quite abruptly. It was closed by some very firm substance, in connection with which some tough bands, with interstices between them, extended from before backwards. These conveyed to the touch somewhat the same sensation as when the finger is passed over the *musculi pectinati* of the heart. As the testis had not descended on the right side, the only conclusion that could be arrived at was, that the mass which obstructed the upper part of the inguinal canal was, in fact, the testis of that side; and the depth at which it was placed may account for a truss having been worn over it without inconvenience.

The explanation of the mode in which two distinct ruptures found their way into a single sac in this case, I conceive to be as follows: The right testicle had probably lodged at the internal ring. The first hernia had descended in front of the testis; a truss was then applied. The two layers of the sac were thus compressed between the testis behind and

the truss in front. The portion of the sac covering the most projecting portion of the testes would thus be subject to a greater degree of pressure than any other portion. I conceive that adhesion between the two opposed layers of the sac had here taken place; but that where, in consequence of the oval shape of the testis, there was a less degree of pressure, no adhesion had occurred. The two layers of the sac would then be united opposite the most projecting part of the testicle, and an aperture would be left on each side of the adhesion thus formed. Whether or not this be the true explanation, there can be no doubt that very firm adhesions existed at the neck of the sac, and that on each side of these adhesions a hernia came down. The stricture at the neck of the first hernia was not so tight as at the neck of the second, so that an impulse was communicated to the first hernia, while the second was really strangulated.

When these two ruptures were down together, they would naturally become more or less distended, and the folds of bowel would only touch each other at one part; one would then displace the other, and this would fully account for the different positions in which the second rupture was found at different times.

The day after the operation all symptoms had been relieved.

ON THE STATE

OF THE

OPTIC NERVES AND RETINÆ

AS SEEN IN THE INSANE.

BY

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Received Dec. 4th, 1867.—Read Feb. 25th, 1868.

IT is not my intention to delay the Medical and Chirurgical Society with any remarks beyond the matter in hand. I have made a series of observations upon the optic nerves and retinæ as seen in the insane, and I wish to confine myself simply to the record of my observations, and to such conclusions as these may seem to justify.

In some instances I find an abnormal state of the nerves, in others a normal state. The cases in the former category may, perhaps, have more interest for the Society; but I may be allowed to say that the absence of morbid change in any one instance is as much to the point as is its presence in another. I submit that the records of negative are as valuable as the records of positive instances, the presence of a healthy optic nerve in one case of insanity being a fact as positive as the discovery of a failing nerve in another case.

I think my records show, to some extent, in what kinds of brain disease, accompanied by insanity, we may expect to find optic changes, and in what kinds we may not expect them. The subject which I have ventured to take in hand is, however, one of extreme complexity, and I would be understood as speaking throughout with great hesitation.

My experience of changing optic nerves and retinæ is, indeed, now somewhat considerable, and many of my observations were repeated twice and even three times. I believe, therefore, that my notes may be received as trustworthy. My conclusions, on the contrary, must be both given and received with great hesitation. I shall be satisfied if my cases, which, comparatively speaking, are few, and my conclusions, which must, therefore, be very fallible, may be taken as stepping-stones to a better knowledge.

The Society will readily understand how an observer, who, like myself, had given some attention to cerebro-spinal diseases, was led on to inquire into the phenomena of insanity.

Even with the present more positive methods of dealing with disease, we scarcely realise sufficiently that a hospital for lunatics is, speaking generally, a museum of cerebral diseases.

Functional or organic disorder of the hemispheres depends upon many kinds and many degrees of change; it may depend upon primary disease of the hemispheres themselves, or upon interference with them by means of disease in other near or more distant parts.

It may, therefore, be objected to me that I have busied myself with an artificial family of disorders, composed of cases so heterogeneous as to forbid any discussion of them in common.

It may be said that I ought to have taken a series of cases classified, not by accidental disorder of a particular function, but by the pathological characters or the original positions of morbid changes.

I might reply by several answers. I would say, however, not by way of answer, but by way of explanation

of my efforts in this direction, that I first examined the eyes of general paralytics for the chance of finding in the retina some of those changes of or about the blood-vessels, which several observers have described in the brain in persons who have died of that disease.

In this I was disappointed, but I was thus led to the discovery of a certain condition of the optic nerve in general paralysis which I shall describe presently. Having in this way begun my researches, I determined to continue them, because the tendency of researches of this kind is to remove the study of mental diseases from the metaphysical methods, and to place it upon a positive basis. I will only now say farther, that I hoped and still hope to find in the indications of the ophthalmoscope some test by which the alienist physician may sift the unwieldy mass of diseases with which he has to deal, and may approach a more certain knowledge of the various pathological conditions of his patients. I hope, for instance, that in dealing with mania the physician may be helped by the ophthalmoscope to decide, in doubtful cases, whether he has to deal with a mania depending upon organic or only upon functional changes, as the phrase is. It seems farther possible that the state of the vascular parts of the eye may indicate to some extent the state of the cerebral circulation in mania.

I shall not spend the time of the Society by entering upon any special questions concerning the nature or origin of mental disorder, nor even concerning its connection with various well-known pathological changes. I shall, for convenience, use the commonest classification, and, perhaps, the most useful, viz. general paralysis, idiocy, dementia, mania, monomania, and melancholia. I shall make a separate class of cases of insanity depending upon or connected with epilepsy. Such a classification is, I know, logically indefensible, but it will serve our purpose sufficiently well.

My observations have been made partly in the West Riding Lunatic Asylum, at Wakefield, with the help of Dr. Crichton Browne, whose diagnosis of each case I place in my schedules; and partly in the North and East Riding Asylum, at Clifton,

near York, with the help of Dr. Christie, who selected and named for me a number of well-marked cases. I cannot sufficiently thank these gentlemen for their courtesy and pains. Upon Dr. Browne's time and patience I made large and repeated demands, to which he most generously responded.

The examinations were made at various dates during the second half of the year 1867.

The examination of the eyes of lunatics is a very tedious and often a very trying task. My natural anxiety to note down no observation of a doubtful character, while, at the same time, to observe was so difficult, would have been too heavy a strain upon my attention had not Dr. Browne and Dr. Christie so materially lightened my labour by their interest in my work and by their hearty aid.

I shall draw the attention of the Society, in the first place, to my cases of general paralysis.

It is in this form of insanity that I have the most definite results to communicate.

GENERAL PARALYSIS.

Cases of especial interest—4, 8, 10, 11, 13, 14, 16, 19, 21, 28, 29, 35, 37, 38, 42, 47, 49, 52.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	William Brear, æt. 36, Wakefield, W. R. L. Asylum	First stage. Second examination (four months later), in the first stage yet, being slow progress	Rather white. Second examination (four months), as before; disc cannot be called decidedly atrophied	First and second examination, a curious instance of choroidal exudation with ramifying vessels, like Liebreich's 'Atlas,' tab. iv, fig. 5. This patch extends from the region of the spot almost up to the disc.
2	William Walker, æt. 37, Wakefield Asylum	Second stage	Normal? Both discs are much smaller than natural. Hypermetropia?	Normal?
3	Thomas Wroe, æt. 46, Wakefield Asylum	Second stage	Too white Veins somewhat large.	Too white.
4	William Snead, æt. 49, Wakefield Asylum	Beginning of third stage	Commencing white. Atrophy distinct	Complete white. Atrophy.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
5	Richard Webb, æt. 46, Wakefield Asylum	First examination, second stage Second examination (three months later), end of second stage or beginning of third	First examination, rather white Second examination, as before	Healthy? Second examination, healthy?
		Post-mortem examination.—Membranes thickened. Arachnoid attached to pia, and many vascular connections. Sulci gaping; grey matter shallow and light coloured, in some places scarcely distinguishable from medullary. Many punctæ sanguineæ on section. Medullary substance very watery and glistening. Ventricles capacious and filled with serum. Weight of whole brain 43½ oz.; of cerebellum, pons, and medulla, 6 oz. Optic nerves unfortunately not noted.		
6	Joseph Sykes, æt. 40, Wakefield Asylum	Incipient Second examination (in four months), first stage, slow progress	Normal (Perhaps a little coarsely pink. Second examination, normal	Normal. Pupils very contracted.) Second examination, disc decidedly larger and more staring than right.
7	Henry Cartwright, æt. 41, Wakefield Asylum	Second stage, far advanced	First examination, both discs atrophic and retinal vessels fine. Second examination (six weeks afterwards), previous observation verified.	
8	Duke Waddington, æt. 49, Wakefield Asylum	Second stage	Both discs atrophic, very decidedly; vessels fine; pupils very contracted.	
9	James Greenwood, æt. 45, Wakefield Asylum	Second stage Second examination (in three months), much the same.	Both discs rather atrophic; retinal vessels fine. Second examination, disc atrophic, not far advanced.	Second examination, scarcely to be called atrophic, but vessels rather large and plum coloured, and some of them a little tortuous.

10	Louisa Lockwood, æt. 38, Wakefield Asylum	Beginning of second stage	First examination, apparent outer edge indistinct and pink; opposite edge too white Second examination (four weeks after), appearances the same	First examination, edge clearing all round; decided atrophy at apparent *inner*. Second examination, decided atrophy all round the disc.
		N.B.—For illustration of this and all similar cases *vide* Liebreich's 'Atlas,' tab. xi, figs. 8, 9.		
11	Ann Taylor, æt. 39, Wakefield Asylum	Second stage	First examination, no change that can be decidedly noted; doubtful Second examination, appearances still doubtful; disc seems too pink and edges not clear	First examination, disc pale, atrophic? vessels seem too fine. Second examination (in six weeks), I am now sure that there is atrophy of this disc.
12	Sarah Maccaby, æt. 46, Wakefield Asylum	Second, passing into third stage Has had several attacks of "apoplectiform congestion."	First examination, ocular disc an unnatural colour, being exsanguine and slaty grey in colour; edges indistinct, and little patches (of old infiltrations?) about the borders Second examination (in four weeks), disc whiter	First examination, commencing atrophy. Second examination, I find noted "decided white atrophy." (This eye seems to be blind.)
		Autopsy.—Brain very much wasted and atrophied *generally*; pia closely adherent to pale and shallow cortical substance; ventricles large; and quantity of serum escaped.		
13	James Nowlin, æt. 45, Wakefield Asylum	First examination, beginning of second stage	First examination, discs do not seem quite natural; pupils, however, much contracted and not sufficiently dilated by the atropine.	
		Second examination (six weeks later), beginning of third stage	Second examination, nutrition of nerves decidedly impaired; edges indistinct, and discs of a dirty or slate colour.	

	Name, place of observation, and age.	Name and extent of disease.	Right eye.	Left eye.
14	Jonathan Allen, æt. 41, Wakefield Asylum	First examination, second stage	First examination, both discs suffused pink; edges only to be made out on very careful observation; vessels natural size; pupils very contracted.	
		Second examination, beginning of third stage	Second examination (in six weeks), discs both decidedly whitening; edges in some places becoming sharp.	
15	John Hyde, æt. 37, Wakefield Asylum	Second stage	First examination, a great many vessels converging to both discs, especially to this; edges a little indistinct; no decided change however	As right, but less marked; might pass for a normal appearance.
		Second examination, advancing in second stage	Second examination, gives no more positive results.	
16	Peter Reynolds, æt. 48, Wakefield Asylum	Second stage	Both discs scarcely distinguishable from retinæ, except by convergence of vessels; pink, and edges barely to be made out.	
		Second examination (in two months), is still to be called in second stage, but much worse	Second examination (in two months), much ocular ataxy; very decided suffusion and *slight* exudation on and about both discs; edges invisible; both discs much affected and vessels many, but right the worse.	
17	John Benson, æt. 35,[1] Wakefield Asylum	Beginning of second stage	First examination, commencing simple atrophy	As right.
			Second examination (four weeks after), first observation verified	
18	John Plumer, æt. 43,[2] Wakefield Asylum	Second stage	Simple atrophy, not extreme, but decided	Simple atrophy, as right.

[1] 17. J. B. is now advanced in the third stage, and his sight is very much impaired.
[2] 18. J. P. Vision much impaired before death.

19	Goodworth Fox, æt. 33, Wakefield Asylum	First examination, second stage	Edge pink and invisible towards apparent outer side; atrophic and sharply defined on opposite side	An excess of pinkness and vascularity on apparent outer edge, but not to be pronounced unhealthy.
		Second examination (one month later), end of second stage	Second examination, this disc clearing into simple atrophy	Second examination, as before.
20	James Birkin, æt. 34, Wakefield Asylum	Incipient	Discs apparently normal, but not to be seen very well, the patient being very unmanageable.	
21	Mary Ann Sykes (21 ward), æt. 37, Wakefield Asylum	First examination, second stage	Vessels rather large and dark coloured, and disc pinker than natural	Vessels rather large and dark coloured; disc decidedly whitening.
		Second examination (in two months), third stage	Second examination, vessels in both discs diminished, and nutrition of both much impaired; they are slaty grey, with ill-defined edges (*vide* Liebreich, tab. xi, fig. 7).	
22	William Hanson, æt. 40, Wakefield Asylum	Incipient general paralysis	Retina very anæmic; disc natural	Retina anæmic; disc said by self and three independent observers to be decidedly whiter than right disc, but cannot be called decidedly atrophied.
23	Edward Thornton, æt. 35, Wakefield Asylum	Third stage and death	Complete amaurosis some weeks before death.	
24	John Carrodus, æt. 36, Wakefield Asylum	Third stage and death	Complete amaurosis.	
25	William North, æt. 63, Wakefield Asylum	Third stage and death	Amaurosis nearly complete, but can tell light from darkness.	

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
26	Thomas Binks, æt. 43, Wakefield Asylum	Well on in second stage	Disc as pink as retina; edges unseen; disc found only by convergence of vessels	Decided commencing atrophy; edges not even, but rather angular, not jagged, are sharply defined.
27	James Henry Winn, æt. 28, Wakefield Asylum	Second stage	Disc pink as retina, distinguishable only by entrance of vessels (no infiltration ?); pupils very contracted	As right.
28	Thomas Kay, æt. 46, Wakefield Asylum	Second stage far advanced	Marked atrophy, invading from apparent inner edge; indistinctness and pinkness at opposite edge	Disc quite atrophic, white; edges clean cut; vessels fine.
29	Mark Longden, æt. 34, Wakefield Asylum	First stage far advanced	Disc too white; edges clean cut; vessels beautifully designed upon the disc	Whiter still; extreme atrophy, or at any rate fully developed.
30	Mary Menshull	Incipient general paralysis; progress very slow	No marked change in either disc.	
31	Abraham Whittaker, æt. 49, Wakefield Asylum	First stage	Must be called normal, though it is a little suggestive to me of commencing atrophy	Disc quite atrophied, but is surrounded by irregular patches of (syphilitic ?) choroiditis.
32	William Woodhead, æt. 41, Wakefield Asylum	Beginning of second stage	Commencing atrophy; congestion towards apparent outer edge; other edge whitening	As right, but more distinct.

33	Harriet Brown, æt. 46, Wakefield Asylum	End of second stage, slow progress	No pronounced atrophy; disc a little staring? a small hæmorrhage in the course of a vessel bending round the yellow spot	Is decidedly whiter than right, but neither disc can be certainly called atrophied.
34	William Robinson, æt. 62, Clifton Asylum	End of first stage	Marked atrophy; nerve slaty grey; vessels fine; edges clean cut, but not very even	As right; simple atrophy.
35	James Butler, æt. 44, Clifton Asylum	First stage	Disc very pink, and edges, especially (apparent) outer, indistinct. I should not pronounce it decidedly changed except for the condition of left disc	Decidedly whiter than right disc, especially at apparent inner edge, the others being still pink. No doubt right is congested and left whitening.
36	Richard Davis, æt. 32, Clifton Asylum	End of first stage	Opaque (traumatic)	Disc decidedly too white, but not completely atrophied. N.B.—There is some little myopic cupping, but not enough to confuse the diagnosis.
37	Michael O'Connor, æt. 55, Clifton Asylum	Second stage	Disc in congestive stage, very pink, and many converging vessels; edge dim, especially at (apparent) outer side	Disc beginning to whiten; very different to right disc; edges staring, except some dimness at (apparent) outer; about tint of natural nerve at that part.
38	Charles Mercer, æt. 30, Clifton Asylum	First stage, in arrest, and seeming quite well	Decided commencing atrophy, with several small tortuous vessels	A little white crescent or "arcus" on (apparent) outer edge, but I should not have noted this save for appearances in right.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
39	William Kirton, æt. 37,[1] Clifton Asylum	First stage, well marked	Some slight indefinition about the edge of the disc	Healthy.
40	— Querchet. Recorded by M. Bouchut, 'Du diagnostic,' &c., p. 333	Stage not stated, probably the second	Atrophy of optic disc	As right, and with central excavation; not glaucomatous, but deeply bowl shaped.
41	William Ledsone, æt. 40, Wakefield Asylum	First stage, but rapidly progressed to death	Commencing atrophy of disc	As right, but still more marked.
		Post-mortem examination.—Pia and arachnoid thickened. Brain surface watery and hollowed out into deep sulci and depressions, one especially in course of middle meningeal artery. Grey matter pale and shallow. Medullary very rosy and speckled and marbled with rosy patches. Choroid plexus pale and anæmic. Atheroma of basilar artery and other trunks.		
42	Walter Griffiths, æt. 44, Wakefield Asylum	Third stage, near termination in death	Disc atrophied; vessels natural size, but varicose; edges not "punched out," but obscured by some recent effusion. (Patient blind.)	As right, but there were *no vessels visible on the surface*; edges as right.
		Post-mortem examination, January 12th, 1868.—Five ounces of serum escaped on removing the brain. Arachnoid slightly thickened and opaque near median fissure. Brain soft and watery. Cortical substance pale and shallow. Convolutions shrunk. Sulci gaping. Numerous and persistent punctæ sanguineæ. Large effusion into ventricles. Corpora quadrigemina wasted and softened, rounded outline lost, testes being quite wasted and almost diffluent. Optic nerves, tracts, and commissure all wasted and attenuated, and semitransparent, of a pale-pinkish, bluish-grey colour. The wasting is more decided as the tracts are traced backwards. Olfactory nerves wasted almost away.		

[1] In a letter I lately received from Dr. Christie, telling me that the patients I examined at Clifton (Nos. 34—39) were all living, he adds that he has ascertained some impairment of vision in Nos. 34, 36, 37, 38, 39.

43	Samuel Blamire, æt. 35, Wakefield Asylum	Second stage	Patient very unmanageable, and inspection very difficult. Disc thoroughly disorganised, having something the appearance of "optic neuritis," but not well seen	Disc dusky and no doubt abnormal, but very hard to see.
44	Charles Redfearn, æt. 39, Wakefield Asylum	Beginning third stage	Disc dusky at apparent outer edge, and lots of tortuous vessels; the other edge whitening. This disc is in an earlier stage than left disc	Contrast very interesting; apparent inner half of disc quite pearly; the other half is traversed by a few fine vessels.
45	John Rayner, æt. 51, Wakefield Asylum	First stage. At commencement under Dr. Allbutt, at Leeds Infirmary	Nothing marked in either eye save a little peripapillary congestion, which may or may not be morbid.	
46	Mark Whittaker, æt. 43,[1] Wakefield Asylum	Second stage	Disc decidedly redder than left disc. There may be a little atrophic tendency about some parts of edge of disc	Disc seems a little redder than natural.
47	John Sykes, æt. 34, Wakefield Asylum	Beginning of third stage	Optic appearances most interesting. Both discs large. Disc whitening in a blotchy way towards apparent inner edge, and a white line creeping round half circumference; from the white it shades into a dusky reddish-brown, and so into a fresher pink at the apparent outer edge, which is quite indistinct; edge blurred all round; vessels rather many	Disc whiter; edge more defined; vessels passing brightly and rather numerously over it. The atrophy is scarcely to be called pearly or staring.

[1] 46. M. W. Now advanced in third stage. Vision much impaired. August 24th, 1868.

	Name, place of observation, and age.	Name and extent of disease.	Right eye.	Left eye.
48	William Parkinson, æt. 40, Wakefield Asylum	Beginning of second stage	Were it not a case of general paralysis I should say the discs were healthy, though not quite of healthy look. The discs are alike, but both have a hard pearly look at the apparent inner edges. Two other observers called them white before I had pronounced upon them. Over the white parts are one or two fine vessels, rather too thready and a little curly—these are suspicious.	
49	John Maguire, æt. 60, Wakefield Asylum	Second stage	Optic appearances very interesting, the discs whitening evenly all round, and not from *outer* edge. Disc dirty red or brown and blurred; dirty white ring all round; depth equal half radius; vessels suspiciously *fine*	Disc still more of the muddy red appearance, probably less advanced than right disc.
50	Joshua Wilson, æt. 39, Wakefield Asylum	Second stage	Disc dusky, but not distinctly diseased	Disc reddish-brown; edges indistinct; a good many vessels. It is decidedly and characteristically affected.
51	Patience Kershaw, æt. 40, Wakefield Asylum	Beginning third stage	Discs small and reddish; pigment about edges. Unaffected?	
52	Thomas Brown, æt. 44, Wakefield Asylum	Second stage. Only retains a few words, and curiously unable to receive words; though not deaf, cannot understand what is said to him	Atrophy commencing in a slightly different degree in each eye. In this case the whitening seems of the simple kind, invading from apparent inner edge (and not? preceded by any dusky redness, &c.).	
53	William Henry Smith, æt. 35	Beginning of second stage	Discs perfectly normal.	

I have scheduled 53 cases of general paralysis, in 5 of which I find no change in the optic nerve and retina.

Of the remaining 48, I find atrophy of the optic disks in its various stages in 41 cases, and 7 cases must be marked as doubtful. In all doubtful cases I made two or three examinations at intervals of a few weeks.

I note the following points:

1. That atrophy of the optic nerves takes place in almost every case of general paralysis, and, I may add, of the olfactory nerves also.

2. That it travels slowly down from the optic centres and along the tracts, becoming evident to the ophthalmoscope at about the end of the "first stage."[1]

3. It becomes apparent as a hyperæmia of the nerve with slight exudation, but without much stasis—as a "red softening," in fact. It then whitens, generally from the real outer edge inwards, the nerve becoming white and staring, and its edge sharply defined. (Sometimes it takes a slate colour. *Vide* Liebreich, pl. xi, figs. 6, 7, 8, 9.)

From Case 38 it appears that the smaller vessels become fine and very tortuous before they vanish. If there has been decided exudation, the edges are, for a time, uneven, but the "punched-out" look always establishes itself in the end.

This kind of "white atrophy" may, in general paralysis, be watched throughout with great accuracy. Ophthalmic surgeons are rarely called upon to examine an optic nerve in the earliest stages of its failure, as these stages are not generally attended with marked loss of vision. They are, therefore, more familiar with the latter or white stages. The process I have above described may be called chronic optic neuritis, as opposed to the state commonly known as optic neuritis, a more highly vascular condition with great exudations, passing on into a more ragged and crushed-looking atrophy, which again, however, after a long period of time, may end in the clean-edged form.

[1] Such was my opinion at the time when this paper was read. Since that time I have made a large number of sections of the optic tracts and tubercula quadrigemina in cases of general paralysis, and have been led to the opposite conclusion.

4. The atrophy of the nerve seems to bear no fixed proportion to the ataxy of the orbital muscles seen in general paralysis. This ataxy is probably dependent upon the same causes as the ataxy of the articulating and other muscles.

5. The nerve changes are generally proportionate to the well-known contraction and dilatation of the pupils. These contract in the early or hyperæmic stage, and dilate as white atrophy succeeds.

6. As atrophy of the optic nerves can seldom be surely ascertained in the incipient stages of general paralysis, its diagnostic significance, therefore, is not great. Its value lies rather in its important pathological significance.

Appendix to Cases of General Paralysis.

Dr. Browne has been kind enough to forward to me a report of such autopsies as have taken place upon any of the above cases, viz. upon Nos. 3, 4, 10, 12, 14, 18, 19, 33, 47. In all, the membranes were found thickened and the convolutions wasted and water-logged. In all but two atheromatous disease of the arteries is noted.

In No. 3. The corpora striata, thalami, and corpora quadrigemina were "flattened, as if wasted."

In No. 4. "All the cerebral nerves much wasted; the optic nerves, commissure, and tracts, especially, atrophied."

In No. 14. "The optic thalami flattened, as if wasted."

In No. 18. "Corpora quadrigemina flattened, wrinkled, and softened; cerebral nerves also softened."

In No. 47. "Optic nerves, commissure, and tracts, white, flattened, and wasted."

P.S.—Since this paper was in type No. 26 has died, and, in addition to the usual changes in the membranes and convolutions, the optic nerves were degenerated. Dr. Browne says, "the nerves appeared quite plump and round, and I should have reported them as healthy but for their *white glistening appearance* [white atrophy, T. C. A.]. On examination they were quite without consistence, and were chiefly made up of connective tissue and watery fluid."

Of mania I have noted 51 cases. Of these, the state of the nerves was, in 25 cases, symptomatic of intracranial disease; in 13 cases it was of doubtful meaning; and in 13 cases I found either no changes at all or only local changes, such as glaucoma, myopia, &c. I think the Society will be surprised to hear of symptomatic change being found and suspected in so large a number of cases. It was far more than I looked for. But it will be remembered, on the other hand, that the patients submitted to me were always well-marked cases of disease, and in the larger proportion of them organic disease was suspected on grounds independent of my conclusions. From my list of mania cases I propose the following points for future observation:

1. That symptomatic changes in the eye are to be found in a large proportion of cases of mania.

2. That if cases known to be functional only, or incorrectly named (such as erotomania, transient mania, hysteria, &c.) be omitted, the proportion of cases presenting permanent change in or near the optic discs is still larger.

3. That both in mania depending upon organic causes and in functional mania the back of the eye, if observed within a few days after a paroxysm, presents a vascular suffusion or pinkness, a pinkness so great after severe paroxysms as to obscure the disc. No exudation is seen in these cases, unless there exist some permanent mischief.

4. That during the paroxysm, on the contrary, the disc is anæmic, perhaps from spasm of the vessels.

5. That the permanent changes in the disc are due either to stasis from obstruction to the intracranial circulation, with consecutive atrophy; or to *ramollissement* ending in simple white atrophy; or they may present changes of a mixed character.

I may make a few further remarks upon my propositions concerning the hyperæmia or blush of the optic disc and retina in mania. I found this so frequently in maniacs who had recently passed through a paroxysm that I began to think I could detect cases of mania by this appearance only. However, I carefully avoided any theorising during my inves-

tigations, lest I should unwittingly vitiate my observing powers. On reading my lists over, however, it occurred to me that this suffusion, which I also saw sometimes upon the conjunctiva, might be due to a paralysis of the vaso-motor nerves.

I instantly turned to the notes of a case which I had by good luck been able to observe during a paroxysm, a piece of rare good fortune.

The Society will see that I have there noted an anæmic retina, and a whitish state of the disc which I put down to commencing atrophy, though with some doubt. May there be in the eye, and so in the brain, a spasm of the arteries—an epilepsy of the mental functions—followed by a paralytic dilatation of longer or shorter duration? Of course my one case can be nothing more than a suggestion of such a mode of action. The appearances of excessive vascularity seemed to last, on an average, from five to eight days. I watched its fading in several cases.

MANIA.

Cases of especial interest—2, 4, 10, 12, 22, 24, 25, 26, 27, 28, 31, 35, 38, 39. *Mania after rheumatism*—51.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	John Kendall, æt. 44, Wakefield Asylum	Mania passing into dementia	Simple white atrophy, with even edges	As right.
		Autopsy.—Chronic degeneration of brain.	This patient is quite blind.	
2	W. Beaumont, æt. 48, Wakefield Asylum	Chronic mania; subacute meningitis? Three months afterwards I hear has had clot on left side, probably in the hemisphere. Previous, therefore, perhaps softening	Disc atrophied, edges patchy	Disc atrophied; edges irregular, and patches about and near them, probably of old exudation.
		Autopsy.—General wasting and disorganization of brain; atheroma of vessels; weight of brain 42½ oz. This patient died after an apoplectic seizure.		
3	Kate Kavanagh, æt. 17, Wakefield Asylum	Hysterical mania; severe case This patient completely recovered	Discs both healthy, but perhaps a little too pinky; certainly pinker than average	
4	Walter Lee, æt. 14, Wakefield Asylum	Three or four attacks of recurrent mania; meningitis Discharged recovered	Disc suffused; vessels large, plum-coloured, and numerous	As right.
			Certainly some obstruction to the circulation of the retina.	
5	Henry Brown, æt. 45, Wakefield Asylum	Chronic mania	Cornea opaque	Disc, &c., natural.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
6	Jos. Hall (No. 1 ward), æt. 44, Wakefield Asylum	Chronic mania; partial paralysis. *Autopsy.*—General wasting of brain; weight 42½ oz.	Discs rather white at the edge, but probably healthy.	
7	Joseph Hall (No. 2 ward), æt. 75, Wakefield Asylum	Chronic recurrent mania, passing into senile dementia	Disc atrophic; consecutive atrophy, with ill-defined edges	As right.
8	Benjamin Holt, æt. 60, Wakefield Asylum*	Chronic mania	Vessels large, and some tortuous; disc too white	Vessels large; disc too white; no infiltration.
9	William Brown, æt. 47, Wakefield Asylum	Chronic mania; constant excitement four months; chronic meningitis	Normal? Appearances doubtful	Outline indistinct; some infiltration; vessels well marked, but not much swollen.
		Autopsy.—Wasted brain; weight 43 oz.; atheroma of vessels; gritty excrescences, rough and hard, on upper surface of corpora quadrigemina; pia mater glued to layers of arachnoid, but separable from brain. Death occurred during an apoplectic fit.		
10	George Crossley, æt. 34, Wakefield Asylum	Convalescing from a most violent attack of mania. Now has great disturbance of vision, flashes of light, &c., also visual hallucinations	Very deeply congested; vessels many and full; disc only to be recognised by entrance of retinal vessels, where there is a white spot	As right, but even more marked in degree.
11	Thomas Camm, æt. 18, Wakefield Asylum	Mania and morbus cordis	First examination, slightly congested Second examination (six weeks later), right as before.	First examination, photophobia. Second examination, still some photophobia, but disc seen, which is more congested than right disc.
12	Joshua Kitson, æt. 35, Wakefield Asylum.	Mania; chronic meningitis Died some months subsequently.	Atrophied; edges not clean cut, nor yet very irregular, but ill-defined Pupils contracted.	Almost as right; no important difference.

* Same case as No. 26. Examined at an earlier date.

		At autopsy.—Diagnosis verified; thickening and opacity of arachnoid; adherence of membranes; serous effusion; capillary congestion; tubercle in lungs.		
13	Jane Turnpenny, æt. 21, Wakefield Asylum	Bad case of "erotomania"	Discs, &c., both normal.	
14	John Spencer, æt. 23, Wakefield Asylum	Mania functional; subsequent complete recovery	Retinæ anæmic; discs healthy.	
15	Daniel Milligan, æt. 53, Wakefield Asylum	Mania; subacute meningitis	Disc very pink, not to be distinguished in colour from the retina. Central veins well marked and plum-coloured. Localised pigmentary retinitis.	As right, perhaps a little more marked.
16	Fred. Simpson, æt. 35, Wakefield Asylum	Acute mania (commencing Graves' disease?)	There is a slight change in the appearance of both discs, but signifies, probably, commencing glaucoma.	
		Autopsy.—Brain wasted; weight 43 oz.; pia mater and arachnoid thickened, opaque, and adherent to surface of brain. Symptoms of general paralysis set in subsequent to examination.		
17	William Denton, æt. 38, Wakefield Asylum	Chronic mania	Retinæ anæmic; discs normal.	
18	James Richmond, æt. 34, Wakefield Asylum	Recovered from an attack of acute mania	No decided change in either eye; perhaps some fulness of vessels, which are rather plum-coloured.	
19	Joseph Broughton, æt. 52, Wakefield Asylum	Chronic mania and meningitis	Disc appears abnormally white, but very transitory view obtained	Could not be seen; patient very difficult to manage.
20	John Beckwith, æt. 42, Wakefield Asylum	Chronic mania	Retinæ anæmic; discs normal.	
21	Harriet Lee, æt. 39 (*vide* Walter Lee, No. 4, her son), Wakefield Asylum	Recovering from mania. (Has also slight Graves' disease?)	Vessels very tortuous and numerous; disc veiled by infiltration	As right.
		The state known as "optic neuritis," and clearly not the retinal stasis sometimes seen in Graves' disease.		

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
22	Charles Chadwick, æt. 53, Wakefield Asylum	Recurrent mania, slight, right hemiplegia	Decidedly atrophic, edges clean cut	Atrophied; a brilliant white nerve.
23	Martin Devine, æt. 35, Wakefield Asylum	Recurrent mania	Back of the eyes presents marked changes, but they are the changes due to myopia.	
24	Michael Wouldhave, æt. 55, Wakefield Asylum	Recurrent mania, no doubt caused by organic disease; attacks very violent	Commencing atrophy; apparent outer edge vascular and suffused, opposite edge whitening and distinct; no infiltration	Complete atrophy; edges distinct, not ragged.
25	Robert Hazelgrove, æt. 23, Wakefield Asylum	Recurrent mania, often very severe. Is now recovering from a violent attack; he is also congenitally imbecile	Too many vessels, which are also plum-coloured; some indefiniteness of edges	Commencing atrophy; edges too clean; vessels numerous; a minute punctiform hæmorrhage near the yellow spot.
26	Benjamin Holt, æt. 60, Wakefield Asylum*	Chronic mania; right hemiplegia; attack two months ago, followed by profound coma	Markedly diseased; atrophy at apparent inner edge, and suffusion and vascularity of opposite edge	As right, differing but slightly in degree.
27	Wm. Williams, æt. 16, Wakefield Asylum	Mania transitoria, functional; attack now passing off	Both discs very pink, and vessels full.	
28	William Broadhead, æt. 43, Wakefield Asylum	Chronic mania, with degeneration of brain	Edges indistinct all round, and disc itself as pink as the retina. Commencing atrophy?	No decided change; vessels a little dark and full.

* This is the same case as No. 8. Hemiplegia had occurred between the examinations.

29	W. F. Seymour, æt. 45, Wakefield Asylum	Mania recent; just admitted	Retinæ anæmic; the discs, however, are very pink, but they are both *exactly* alike, and probably, therefore, healthy.	
30	Ephraim Wood, æt. 37, Wakefield Asylum	Convalescing after acute mania. Gangrene of foot	Disc seems a little white, but cannot be called diseased	Decidedly whiter even than right, but I should scarcely like yet to pronounce it atrophied.
31	B. W. Skelton, æt. 23, Wakefield Asylum	Acute mania (severe). By good fortune we secured an examination during a paroxysm	Retina anæmic; disc rather white, looking like commencing atrophy	As right, except that at one point of the edge of the disc there seems to be some vascularity.
32	L. Henry, æt. 25, Wakefield Asylum	Mania	Retinæ anæmic; discs healthy.	
33	James Grey, æt. 40, Clifton Asylum	Chronic mania, with great depression in intervals of excitement	Disc white and much encroached upon by a patch of pigment; some vessels tortuous; probably some local (perhaps congenital) condition	Healthy.
34	Sam. Jackson, æt. 51, Clifton Asylum	Chronic mania, now passing into dementia	Natural	Irregular in outline and whiter than right disc. No remains of any old exudations.
35	Jos. Blackburn, æt. 29, Clifton Asylum	Mania, probably functional; has been very violent; has been tolerably quiet for three days	Much pinkness of and about disc; vessels full	As right, but even more distinctly marked.
36	Sarah Walls, æt. 64, Clifton Asylum	Chronic mania	Discs, &c., natural.	
37	Thos. Ward, æt. 44, Clifton Asylum	Chronic mania, passing into dementia	Both discs, &c., healthy.	

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
38	Thos. Brighton, æt. 29, Clifton Asylum	Mania violent, with left hemiplegia	Before I was aware of the left hemiplegia I noted that this right disc was much congested, and the retinal vessels many, full, and plum-coloured (obstruction)	Natural.
39	W. G. Broadbent, æt. 36, Clifton Asylum	Old and bad case of mania. No evidence of organic disease; now quite quiet	Disc red, and vessels rather many and full	Even more congested than right, the edges of the disc being almost indistinguishable; no exudation.
40	Mary Wright, æt. 23, Clifton Asylum	Old history of mania and of states resembling catalepsy, but not very well marked; probably organic disease	Commencing atrophy; some vessels tortuous; apparent inner edge very white	As right, but a little whiter.
41	Mary Bellerby, æt. 46, Clifton Asylum	Chronic mania; seldom excited	Healthy	Healthy.
42	Isaac Hutton, æt. 40, Clifton Asylum	Mania; supposed syphilitic meningitis	Slight atrophy, apparent inner being white; edges defined	More marked still, but far, however, from being complete; edges a little ragged.
43	Thos. Kelley, æt. 26, Wakefield Asylum	Chronic mania	Disc pinkish, more than natural; some excess of vessels	Cornea opaque.
44	Wm. Bedford, æt. 67, Wakefield Asylum	Recurrent mania (degeneration of brain ?)	Discs, &c., both normal.	

45	Jos. Morris, æt. 37, Wakefield Asylum	Mania; paroxysm recently passed by	No marked change in either eye, but right disc whiter than left.	
46	Edw. Rayner, æt. 48, Wakefield Asylum	Chronic mania; subacute meningitis	Disc whitish at apparent inner edge, and some pinkness about the opposite edge, but taken alone should not be pronounced diseased	Disc very different to right disc, being whiter at apparent inner edge and more suffused about opposite edge. Taking the two discs together, I have no doubt of progressive change.
47	Wm. Woodhead, æt. 41, Wakefield Asylum	Mania; chronic meningitis	Disc ill seen, as the patient was very violent, and kicked and spat while being examined. But the vessels are dark and large, and there seems to be effusion, or, at least, much congestion	Disc ill-seen, as was the case with right. But the disc and neighbouring retina look cloudy, and the vessels near the disc are somewhat obscured, though not concealed. No doubt this disc will atrophy.
48	Rob. Feathers, æt. 21, Wakefield Asylum	Acute mania, probably functional; attack passed by some few weeks ago	Disc red or muddy, and with whitish edge at apparent inner side. But it may be nothing more than hyperæmia (a doubtful disc)	As right, but less in degree; not quite right in appearance; doubtful.
49	John Cockcroft, æt. 44, Wakefield Asylum	Chronic mania; degeneration of brain	Discs both quite normal.	
50	Henry Wickham, æt. 25, Wakefield Asylum	Recurrent mania, hereditary	Disc small and reddish	Disc whiter than right, and edges indistinct.
51	Abraham Waddington, æt. 35, native of Cononley. Attended by Dr. McNab, of Crosshills. Wakefield Asylum	Admitted with violent mania, sequence of rheumatic fever; is now recovered (Feb. 18, 1868); has tubercle of lungs	Neither can be pronounced diseased. White atrophy *distinct*, though not very extreme; can read No. 6 Jäg. with difficulty	Disc atrophied, but not so decidedly as right disc; reads No. 3 Jäg. with difficulty.

The next portion of my schedule contains 38 cases of *dementia* not connected with epilepsy. Most of them, however, were due to organic disease in the brain, and were picked out for their severity.

Of these I found disease in the optic nerve or retina in 23 cases; I found 6 in a doubtful condition, and 9 were healthy. It will be seen that in simple acute dementia (uncomplicated with organic disease), however profound, no changes in the optic nerve are recorded.

I will only note further, that in dementia, where atrophic changes were seen by the ophthalmoscope, I both made inquiries concerning the sight, and, in some cases, tested it. I confirmed my former belief, that statements concerning visual power, whether made by patients, or by their friends, are of no value (*vide* Dementia, *e.g.* No. 29); and, moreover, that failure of sight, as tested by types, is wholly an oculist's symptom, and bears little proportion to the amount of atrophic or other disease that may be seen in the nerve. Mapping of the field of vision is a most important physician's symptom, but with lunatics this would have been simply out of the question.

DEMENTIA.

Cases of especial interest—1, 3, 5, 7, 11, 12, 13, 19, 23, 25, 26, 27, 29, 35, 38.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	James Dutton, æt. 48, Wakefield Asylum	Profound dementia; paraplegia; can but just walk	Marked atrophy of disc	Cornea opaque.
		Autopsy.—Dura mater thickened and puckered; brain wasted; weight 44 oz.; vessels atheromatous, and projecting from cut surface; remains of old small hæmorrhages.		
2	Joseph Sykes, æt. 46, Wakefield Asylum	Dementia	Vessels well-marked; eyes healthy?	
3	Jane Dixon, æt. 65, Wakefield Asylum	Senile dementia; partial paralysis of all limbs	Discs both much changed in outline, and rather too white; frequent pigment spots about margins of both; blotchy condition of both retinæ, especially of *left*.	
4	— Bancroft, female, æt. 24, Wakefield Asylum	Acute dementia	Both discs, &c., healthy.	
5	Jemima Batty, æt. 23, Wakefield Asylum	Acute dementia and bronchocele	Active change in both discs; much congestion for about three fourths of margin in both, which is quite concealed by a veil of exudation; there seems a tendency to whiten at both (apparent) inner edges; vessels many, the *smaller* slightly tortuous in many cases.	
6	Thomas Messenger, æt. 57, Wakefield Asylum	Dementia	Discs both atrophic, not very far advanced; vessels fine.	

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
7	John Kendall, æt. 62, Wakefield Asylum	Dementia following mania	Simple white atrophy, with even edges	As right.
8	Benjamin Kemplay, æt. 62, Wakefield Asylum	Old case of dementia; degeneration of brain	No marked change	Slight, but I think decided commencing atrophy.
9	Henry Oldham, æt. 61, Wakefield Asylum	Dementia; left hemiplegia; old clot	Disc normal? perhaps slightly pink and, it may be, a little indistinct towards (apparent) inner margin	Disc compressed from above downwards, and much whiter than right disc; could not alone be called decidedly atrophic.
10	Nathan Bentley, æt. 38, Wakefield Asylum	Dementia; tubercle	Both discs misshapen and compressed from above downwards; vessels a little too pronounced; discs are both a little too pink, and their edges somewhat indefinite; both, however, are exactly alike.	
11	Patrick Carpenter, æt. 50, Wakefield Asylum	Profound dementia, far gone *Autopsy.*—Disorganization of brain.	Discs both atrophied, and small; vessels fine; both eyes quite alike. This patient is quite blind.	
12	Richard Buckley, æt. 31, Wakefield Asylum	Dementia; right hemiplegia; clot	Right optic disc too white Vessels normal.	Left disc, decided atrophy; edges even.
13	Joseph Hall, æt. 75 (of No. 2 ward), Wakefield Asylum	Senile dementia following chronic recurrent mania	Atrophic disc, with irregular edges	As right.
14	Caroline Brooke, æt. 44, Wakefield Asylum	Dementia; chronic disorganization of brain	Vessels rather too pronounced, and some a little tortuous	Disc decidedly too white; atrophic.
		This patient was examined again some weeks later—observation verified.		

15	Benjamin Lister, æt. 49, Wakefield Asylum	Dementia; softening of brain; paralysis of left side from old clot	Healthy	Cornea opaque.
16	William Pepperdy, æt. 42; Wakefield Asylum	Dementia; clot; right hemiplegia; no aphasia.	Healthy	Disc slightly but, I think, decidedly atrophic; decidedly, at any rate, whiter than right disc.
17	Henry Holloway, æt. 21, Wakefield Asylum	Acute dementia; recovering	No marked change, unless it be some pinkness of the discs and a little photophobia.	
18	David Lambert, æt. 32, Wakefield Asylum	Dementia; masturbation	Discs small and pinkish; vessels a little numerous. I think, however, that this is only a presbyopic change, not a symptomatic.	
19	Mary Ann Hessey, æt. 47, Wakefield Asylum	Dementia and Graves' disease. (Comp. Harriet Lee, No. 21 of Mania schedule). Since dead	Disc shows a tendency to atrophy	Left disc decidedly atrophied.
		At autopsy.—Adhesion of dura mater to skull-cap; atrophy of the cortical substance; hardness (sclerosis ?) of the medullary substance; large serous effusions; old pericarditis.	Her sight is known to have been failing for four or five years.	
20	John Maguire, æt. 60, Wakefield Asylum	Old dementia and degeneration of brain	Right disc looks a little atrophied; a pigment ring round one third of circumference on (apparent) inner side	Left disc looks, on the contrary, unnaturally pink.
21	Edward Hurst, æt. 31, Wakefield Asylum	Disease as No. 20, with occasional and various paralyses	Discs both a little troubled, as if with a little former exudation, but no advancing change.	
22	Charlotte Drake, æt. 62, Wakefield Asylum	Senile dementia; disease of capillaries (?), so that the slightest rap upon the arm causes marked ecchymosis, &c.	The media are dull, and the discs and retinæ of a dim muddy look, not exactly anæmic in appearance; no apparent change in the vessels.	
		Autopsy.—Brain soft, wasted, and watery; much atheroma of vessels.		

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
23	John Emmott, æt. 19, Wakefield Asylum	Dementia; profound anæmia	Disc decidedly atrophic at (apparent) inner edge; apparent outer obscured and too vascular	Disc decidedly but not extremely atrophic.
		Autopsy.—Brain substance generally pale; layers in cortical matter well defined; veins and sinuses much distended with blood; all the veins about the base remarkably full. Phthisis pulmonalis.		
24	Joseph Jubb, æt. 60, Wakefield Asylum	Senile dementia	Pigmentary retinitis both eyes; a large increase of vascular ramifications; discs not decidedly abnormal (whitish ?).	
25	Thomas O'Brien, æt. 22, Wakefield Asylum	Dementia following meningitis	Discs pink; considerable increase of vascular ramifications; the circulation of the discs is decidedly affected.	
26	—— Wakefield Asylum	Partial dementia; clot; left hemiplegia, and some difficulty in swallowing	Both discs congested, and most of the margin (especially at apparent outer edge) of both invisible.	
27	Jacob Rogers, æt. 60, Wakefield Asylum	Dementia, old case; double hemiplegia, with partial recovery; strabismus; several clots, some rather large	Both discs markedly atrophic.	
28	Mich. Bartlett, æt. —, Wakefield Asylum	Dementia; right hemiplegia; aphasia	Disc perhaps a little too white, and, when taken with left disc, may be called slightly atrophic	Disc decidedly atrophic.
29	John Carrier, æt. 48, Wakefield Asylum	Dementia; right hemiplegia; no aphasia	Disc, (apparent) outer edge indistinct and suffused pink; opposite edge quite atrophic	Left disc quite atrophic. Stated by self and others that his sight was in no way failing.

				On examination can only read 16 Jäger with left eye, and cannot read ordinary news print even with right.
30	Ralph Harper, æt. 50, Wakefield Asylum	Dementia; old disorganization of brain	The vessels in and about both discs are fine and few. I have little doubt that atrophic changes are going on in both eyes, left disc being much whiter than right disc; but as myopic changes are also going on, I cannot give a decided opinion.	
31	George Wheatley, æt. 25, Wakefield Asylum	Acute dementia	Discs natural;	retinæ anæmic.
32	William Poppleton, æt. 25, Clifton Asylum	Dementia; no history	Commencing atrophy of disc; apparent outer edge indistinct, and opposite edge white	Decided but not complete atrophy.
33	Edward Farrow, æt. 57, Clifton Asylum	Dementia; paraplegia. (Paralysis below the sympathetic region of cord)	Healthy	Healthy.
34	James Tyman, æt. 42, Clifton Asylum	Dementia; organic disease of encephalon; paralysis of articulation (tongue and larynx); remains of right facial paralysis	Extensive syphilitic choroiditis	Decided but not complete atrophy of the disc; choroid healthy.
35	Joseph Pratt, æt. 51, Clifton Asylum	Dementia; organic disease; degeneration of brain	Disc dirty in colour; edges dimmed in places by exudation, but very partial and slight	Disc, edges obscured by wide areola of exudation; vessels *not* large or plum-coloured, and are hidden as they approach and pass through the exudation region. This is probably *genuine* optic neuritis.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
36	Richard Waddington, æt. 33, Wakefield Asylum	Blow on head over left parietal; concussion; interval of health, then gradual change in affections—1, in emotions; 2, in intellect. Wasting and loss of muscular power; certainly has organic disease of brain	Whitish in centre, but cannot be pronounced to be diseased	Left disc compressed and decidedly too white.
37	Ann Gallaghan, æt. 16, Wakefield Asylum	Profound acute dementia, functional	No marked changes, unless it be some largeness of the vessels.	
38	Dennis Higgins, æt. 65, Wakefield Asylum	Profound dementia; irregular paralysis	Patient quite unmanageable; eyes could not be thoroughly examined; but there was complete amaurosis, and the discs were white.	
		Autopsy.—Changing clots on surface of brain; total atrophy exactly up to median line of left half of fornix; wasting of thalami on both sides, which were hollowed out; attenuation of corpora quadrigemina and deep brown discoloration of the nates; optic nerves seemed unaffected to the naked eye, so also commissure and tracts. Under the microscope, however, sections of them showed great proliferation of the connective tissue and atrophy of the nerve fibres.		

In *melancholia* and *monomania*, which I have tabled together for convenience, the relation of events is changed. As a large proportion of these affections are functional only, so I find, on analysing my list, that of 17 cases, the optic nerve and retina were healthy in 10, doubtful in 4, diseased in 3 cases. Of the *four* marked as doubtful, the case of John Booth (No. 1 of the table), which is noted as possibly a case of commencing atrophy of disc, has since shown symptoms of locomotor ataxy, as Dr. Browne tells me. This event accounts for my suspicion of commencing atrophy. I find in the 3 diseased cases that Dr. Browne notes "atheroma of vessels" in one; in another "probable organic disease;" in the third, "chronic disorganization of the brain."

Alienist physicians will not be surprised to hear that I very frequently noted the presence of anæmia of the retina in melancholia.

MELANCHOLIA AND MONOMANIA.

Cases of especial interest—2, 4, 9, 10, 12, 13.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	John Booth, æt. 49, Wakefield Asylum	Melancholia; has since shown symptoms of locomotor ataxy	Contracted pupils; discs rather white, with frequent circles about edges	As right. I cannot say that either disc is unhealthy.
2	Henry Shirt, æt. 51, Wakefield Asylum	Deep melancholia (moral cause)	Disc *reddish*	As right.
			Probably both healthy?	
3	G. Crowther, æt. 45, Wakefield Asylum	Hereditary melancholia (suicidal)	Discs whitish, but probably only anæmic; a little appearance of pigmentary retinitis.	
4	Stephen Sagar, æt. 60, Wakefield Asylum	Monomania; atheroma of vessels	A good many full vessels, and some a little tortuous; disc seems whitening at edges, which are very distinct	Vessels less full and fewer; edge of ocular disc indistinct, as if from very slight infiltration; disc slate-coloured; tendency of part of edge to whiten.
5	Thomas Pinder, æt. 37, Wakefield Asylum	Syphilophobia and hypochondriasis; has since recovered	Both discs healthy.	
6	Hannah Moorhouse, æt. 37, Wakefield Asylum	Religious monomania, with hallucinations of sight and hearing	Both discs, &c., healthy.	
7	Ann Shaw, æt. 45, Wakefield Asylum	Melancholia; scirrhus of mamma	Profound anæmia of both optic discs and retinæ.	
8	Mark Vause, æt. 54, Wakefield Asylum	Melancholia (long history)	Healthy	Healthy.

9	Eliza Parker, æt. 49, Wakefield Asylum	Melancholia; anæmia and morbus cordis from rheumatism	Disc pink and a good deal of vascularity; edges indistinct	As right.
10	James Longley, æt. 51, Wakefield Asylum	Confirmed melancholia; masturbation; probable organic disease	Pinker than natural (healthy?)	Suffusion of colour and vascularity at apparent anterior edge; commencing atrophy at opposite edge.
11	Patrick Heys, æt. 40, Wakefield Asylum	Monomania of suspicion, with sense hallucinations, especially visual	Disc flattened from above downwards	Disc very decidedly whiter than right disc.
12	Christus Seward, æt. 29, Wakefield Asylum	Melancholia, passing into dementia	First examination, discs not natural, but the appearances probably those of hypermetropia. Second examination, I think there is decided hyperæmia of the disc and retina, not dependent upon any change in the *coats* of the eye.	
13	Thos. MacNamara, æt. 28, Wakefield Asylum	Melancholia; old case. Chronic disorganization of brain	Apparent outer edge of disc indistinct and vascular; decided commencing change; opposite edge whitening	Scarcely any change? quite different to right.
14	John Brown, æt. 36, Wakefield Asylum	Profound melancholia; hereditary	Retinæ *not* anæmic; discs healthy.	
15	Thos. Abbott, æt. 56, Wakefield Asylum	Melancholia; injury to head	Right disc pinker than left disc. Healthy?	A greyish-white spot on the course of one vessel, and a rather larger and very distinct one near the yellow spot not on the course of a vessel. Both are, I think, old hæmorrhages.
16	Benj. Copeland, æt. 46, Wakefield Asylum	Melancholia; pigment in skin all over body	Discs healthy. "Pigmentary retinitis" in both eyes.	
17	Rob. Moorhouse, æt. 35, Wakefield Asylum	Melancholia	Discs and retinæ normal.	

Of *insanity depending upon epilepsy* I bring 43 cases. It may surprise some of my hearers to be told that when compared with general paralysis, mania, and dementia, the proportion of epileptic cases which present symptomatic changes in the eye is small.

I note disease of the optic nerve or retina in 15 cases out of the 43. I mark 9 as doubtful; the remaining 19 showed no diseased change.

It is known, however, by those who have used the ophthalmoscope in cerebral disease that epilepsy alone (not dependent upon organic disease) is not usually accompanied by disease of the optic nerve. It is thought that some change in the vessels of the retina may be seen in epilepsy, and some of my cases seem to bear this out. On a re-examination of the table it will be seen that organic disease was known by unilateral symptoms, or otherwise, to exist in most of the cases in which the optic nerves are noted as diseased.

I should perhaps remind the Society that the general diagnoses and the eye examinations were noted by different persons.

INSANITY WITH EPILEPSY.

Cases of especial interest—13 and 41, 22, 24, 27, 30, 33, 36, 37.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	M. Darwin, æt. 25, Wakefield Asylum	Old epileptic and dementia	Disc a little too pink; edges a little indistinct. No marked change	Venæ centrales very large in this eye; otherwise as right.
2	B. Feathers, æt. 39, Wakefield Asylum	Epilepsy five months; dementia	A little infiltration around the margin of disc	Ocular disc, edges very irregular, and a good deal of infiltration about them, especially toward apparent outer edge.
3	Wm. Hy. Ellis, æt. 3, Wakefield Asylum	Epileptic; enfeebled faculties	Discs, or at any rate right disc, is decidedly white, but patient very unmanageable.	
4	Chas. W. Walton, æt. 33, Wakefield Asylum	Epilepsy; blow on the head and exfoliation; loss of portion of cerebral substance	Apparent outer edge of disc invisible; of suffused and vascular appearance; other edge clean cut and whitening	As right, but less in degree.
5	M. Evers, æt. 32, Wakefield Asylum	Confirmed epilepsy; much ocular ataxy	Disc pink, about the colour of the retina, and difficult to find except by convergence of vessels; edges very indistinct; vessels full, not tortuous	As right, but worse.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
6	Wm. Woodhead, æt. 41, Wakefield Asylum	Confirmed epilepsy with dementia; tubercle in both lungs	Atrophied; edges clean cut	Some infiltration and excess of vessels on apparent outer edge; other edges white; atrophy sharply defined.
7	Elizabeth Crackle, Wakefield Asylum	Confirmed epilepsy and dementia	First examination, healthy	First examination, healthy.
		Examined again after three or four days of excitement	Second examination, healthy	Second examination, healthy.
8	Martha Dickenson, Wakefield Asylum	Epilepsy occasional; partial paralysis of right side; no aphasia	Exophthalmos; disc healthy? Vessels a little large	Exophthalmos preceded that of right eye, but now receded. Disc healthy? Vessels *finer* than usual. Disc a little whitish?
9	Hannah Gates, æt. 23, Wakefield Asylum	Is liable to long series of fits, followed by long coma. No unilateral spasm	Healthy	Healthy.
			Nothing abnormal in vessels?	
10	Sarah Walsh, æt. 55, Wakefield Asylum	Confirmed epilepsy and dementia. Lately, impairment of muscular power	Healthy	Healthy. A small pigment patch, probably congenital, upon retina.
11	Sarah Holdsworth, æt. 20, Wakefield Asylum	Severe (traumatic) epilepsy; profound dementia. Depression 1×2 inches at longest diameters at anterior and upper border of right parietal bone	Disc too pink; edges invisible	Ocular disc natural, unless, perhaps, some slight excess of pinkness.

12	Louisa Sellers, æt. 23, Wakefield Asylum	Epilepsy five years' standing and dementia	There is a whitening at part of margin in both discs, but probably only commencing myopic change.	
13	Charles Wood, æt. 27, Wakefield Asylum	Chronic epilepsy and mania, not meningitis. Second examination, has just passed through a violent epileptic paroxysm; has visual and other hallucinations	First examination healthy; vessels large and full. Second examination. On former examination the vessels were noted as rather full than otherwise, but now are very small, fine, and few	First examination, healthy? Second examination, vessels here also smaller than before, but artery and vein distinctly visible.
			Compare this with Case 41.	
14	— Scholefield, Wakefield Asylum	Epilepsy and dementia; epilepsy from childhood; left hemiplegia from childhood	Disc of irregular form and almond-shaped	As right, but a little whiter.
			Otherwise healthy.	
15	Mary Cooper, Wakefield Asylum	Epilepsy; dementia; phthisis	Vessels a little too distinct, and disc rather too pink. Healthy?	As right.
16	Hen. Bence, Wakefield Asylum	Epilepsy; dementia	Disc pinkish? Retina very anæmic	Disc as right; vessels fuller than in right, and more than usually full; retina very anæmic.
17	Isaac Kendrew, æt. 20, Wakefield Asylum	Continual epilepsy and dementia	Disc atrophic	As right.
18	Thos. Greenwood, æt. 30, Wakefield Asylum	Epilepsy and dementia	Pigmentation about the margins of both discs, which, however, are probably quite healthy.	
19	Chas. Paget, æt. 28, Wakefield Asylum	Severe epilepsy and dementia; anæsthesia and impairment of muscular power	Over vascularity and pinkness of disc, but no decided change	As right.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
20	Edwd. Scholefield, æt. 20, Wakefield Asylum	Epilepsy and dementia; hemiplegia of left side with wasting of muscles	Healthy	Healthy.
21	Jane Sheldon, Wakefield Asylum	Epilepsy and dementia	Healthy	Healthy.
22	Rob. Boasman, æt. 33, Clifton Asylum	Dementia, unilateral (right); epilepsy; no aphasia	Commencing atrophy of disc	Perhaps there may be slight atrophy; the disc has a doubtful whitish look, but is not nearly so white as right.
23	Wm. Eggleston, æt. 22, Clifton Asylum[1]	Mania and epilepsy; *suspicion* of organic disease	Right disc decidedly a little whiter than left	Left is also pale, but I would not assert that either disc is atrophied.
24	Geo. Monkman, æt. 35, Clifton Asylum	Dementia and epilepsy; probably organic disease	Disc congested, and vessels many, but I should not, except for left disc, have attached much weight to it	Left disc is very decidedly congested, and its edges for about half circumference on (apparent) outer side misty. Taking both eyes together there is evidence of change.
25	Joseph Williamson, æt. 22, Clifton Asylum	Epilepsy; dementia; much stupor	Right disc, &c., congested; vessels frequent	Healthy.
26	Rebecca Braithwaite, æt. 36, Clifton Asylum	Mania; epilepsy affecting right side; supposed organic disease	Healthy	Healthy.

[1] No. 23, Wm. E—, died January 24th, 1868, of repeated epileptic seizures. Autopsy forbidden.

27	Hannah Humble, æt. 42, Clifton Asylum	Recurrent mania and epilepsy	Both discs and neighbourhood showing the pinkness and vascularity I have often noted after attacks of mania.	
28	Martha Hutchinson, æt. 27, Clifton Asylum	Epilepsy and mania; no unilateral symptoms	Healthy	Healthy.
29	Thos. Storey, æt. 41, Wakefield Asylum	Epilepsy and dementia	Discs, &c., healthy.	
30	Sam. Jowitt, æt. 31, Wakefield Asylum	Dementia and *unilateral* epilepsy	Commencing atrophy of disc	Atrophy more advanced than right, but scarcely to be called complete.
31	Horatio Edwards, æt. 27, Wakefield Asylum	Dementia and epilepsy	No change	No change.
32	George Lawton, æt. 28, Wakefield Asylum	Dementia and epilepsy	Discs healthy? Several (congenital?) patches in both eyes about their margins.	
33	John P. Halligan, æt. 56, Wakefield Asylum	Eleven years standing dementia and epilepsy, following depression of skull from a blow	Decided atrophy	Complete atrophy. Vision obviously impaired.
34	Fr. Richardson, æt. 19, Wakefield Asylum	Dementia and epilepsy	Discs normal, &c.	
35	Alfred Swaine, æt. 26, Wakefield Asylum	Dementia; epilepsy	Some over-pinkness and vascularity in and about both discs, but no marked change.	
36	Isaac Challoner, æt. 49, Wakefield Asylum	Dementia or epilepsy, with evidence of cerebral degeneration	Commencing atrophy; apparent inner edge pearly; opposite edge suffused, and vessels many and fine	Decidedly whiter than right disc.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
37	Thos. James, æt. 17, Leeds Infirmary	Dementia and epilepsy; unilateral (left) symptoms	Has been under my notice for eighteen months, the dementia slowly creeping on. He had, when I first saw him, congestion and exudation in and about both discs. This is now passing into consecutive atrophy with ill-defined edges, &c. Examination, nine months later, shows the edges clearing and becoming definite.	
38	Br. Dyson, æt. 21, Wakefield Asylum	Dementia and epilepsy	Both discs white, vessels few; some indistinctness of inner edge in both.	
39	Alfred Forbisher, æt. 16, Wakefield Asylum	Congenital imbecility and epilepsy	Disc atrophic at apparent inner edge; many tortuous vessels about opposite edge, but it can be distinguished with $3\frac{1}{2}$", not with 2"	Disc atrophic at apparent inner; opposite edge cannot be distinguished even with 4"; probably some little exudation; one fine and curly vessel crosses the atrophied half.
40	Mary Slater, æt. 21, Wakefield Asylum	Dementia, epilepsy; hereditary; father and sister	Vessels to both discs look rather large and a little tortuous, and the discs are not as distinct as they usually are in normal states.	
41	Eliz. Brown, Wakefield Asylum	Dementia and epilepsy. Has just passed out of a status—a succession of fits and coma	Discs anæmic (or slightly atrophic?) She can read No. 1 Jäger. Two vessels of right disc look a little tortuous.	
42	Alfred Burrows, æt. 22, Wakefield Asylum	Dementia and epilepsy	Vessels very fine, almost like embolism	Disc decidedly whiter than right disc. Anæmic?
43	Geo. Ainley, æt. 44, Wakefield Asylum	Dementia and epilepsy	Discs, &c., healthy.	

My last table is one of *idiocy*.

I examined some idiots because I had formed a suspicion from the few cases which had occurred in my own practice, that atrophy of the optic nerves was not uncommon in idiocy. It will be seen that of 12 cases I note very decided atrophy of the discs in 5, and advancing disease in 1, while 2 may be called doubtful. Whether this be due to encephalic inflammations in childhood, or to whatever causes it may be, I leave to future observers to say.

IDIOCY.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	Alfred Crossley, æt. 21, Wakefield Asylum	Congenital imbecility	No marked change in either eye; perhaps some over pinkness and excess of vessels.	
2	Eugene Slin, æt. 19, Wakefield Asylum	Congenital idiocy	Apparently complete atrophy of both discs, which are clean cut and brilliant; seems to see his way well about, but is very stupid.	
3	Charles Blackburn, Wakefield Asylum	Congenital idiocy	Discs decidedly atrophic, having a brilliant whiteness; observation, however, difficult, as the patient has tremulous iris, tremulous eyeball, strabismus, and is deaf and stupid.	
4	George Talbot, æt. 16, Wakefield Asylum	Imbecility after acute hydrocephalus in infancy	Both discs markedly atrophic; outlines irregular, but not uneven or ragged; central vessels of natural size.	
5	Joseph Ogley, æt. 44, Wakefield Asylum	Congenital idiocy; dumb	Discs natural; retinæ anæmic.	
6	Chas. Thos. Watson, æt. 19, Wakefield Asylum	Congenital idiocy	Right disc pinker than left disc I cannot be sure that there is any progressive change going forward.	Left disc, may be in course of atrophy, is decidedly whiter than right disc; also left disc is much larger than right disc.

7	John Newby, æt. 35, Wakefield Asylum	Congenital imbecility	There may be some atrophic tendency, but neither disc can be called diseased.	
8	Walter John Milner, æt. 26, Wakefield Asylum	Congenital idiocy in highest degree	Commencing atrophy of disc Arteriæ centrales beautifully distinct on the pearly ground, especially in left.	Very decided, if not complete, atrophy of disc.
9	Ann E. Haglewood, Wakefield Asylum	Congenital idiocy	Neither disc healthy, but as there is presbyopic disease apparent, I cannot decidedly say whether there is any atrophic tendency or not.	
10	Joseph Heald, æt. 30, Wakefield Asylum	Congenital idiot	Opaque cornea	Presbyopic changes only.
11	Thomas Wood, æt. 19, Wakefield Asylum	Congenital idiot; very slight case	Discs, &c., healthy.	
12	Walter Newton, æt. 20, Wakefield Asylum	Imbecility	Disc rather congested, whitening toward apparent inner edge; opposite edge indistinct and with appearance of old infiltration obscuring the margin; vessels many and full	As right.

Such are the few remarks which I venture to make upon my observations. The main interest of my communication must still lie in the schedules themselves. To enter upon a study of these in detail would be to detain the Society very unreasonably. I point out the main inferences only, namely, that in old or severe organic disease of the brain and its membranes, whether degenerative, hæmorrhagic, meningitic, or due to tumours, obvious changes in the eye are to be seen in a very large proportion.

That in so-called functional diseases, however profound, such changes are to be found only in a very small proportion.

That atrophy of the disc is perhaps a constant symptom of general paralysis, a fact of the highest pathological importance.

That in functional mania and functional epilepsy, but especially in the former, the variations of vascular tension in the retina and disc may have great interest for the observer.

Finally, I hope I have shown that the account of a cerebral disease cannot be complete if without any record of ophthalmic observations.

NOTES RESPECTING

NON-UNITING FRACTURES.

BY

GEORGE W. CALLENDER, F.R.C.S.,

ASSISTANT-SURGEON TO, AND LECTURER ON ANATOMY AT, ST. BARTHOLOMEW'S HOSPITAL.

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WHAT Mr. Bransby Cooper says respecting certain injuries, "how rarely, even in remote districts, are now to be noticed dislocations remaining unreduced through ignorance of the nature of the injury, or of the means to be employed for their reduction," ought to be applicable to these fractures, for their occurrence is not creditable to surgery. They are most unfortunate cases, especially when the lower extremity is involved.

There is a woman, now æt. 43, on our Coburn fund, whom I see from year to year, who broke her leg in the summer of 1852. The fracture of the tibia was very oblique, extending from its upper third to just above the malleolus. Neither bone united, and she now limps with a fixed ankle. If she tried to flex or extend the foot, the attempted movement displaced the fracture, and caused her excessive pain. The source of this pain was not very clear; however, she learned

to avoid the movement, and her ankle-joint has become stiff from disuse.

The non-union of fractures has been attributed to a great variety of causes, constitutional and local. Mr. Amesbury, however, remarks, "by far the most frequent cause of non-union that I have noticed is want of rest, in consequence of the inadequacy of the plans of treatment which have been employed. I consider this to have been the primary cause in almost all the cases I have examined."

And Dr. Hamilton, in his treatise, writes, "I have myself seen a considerable number of examples of non-union, but in not one of my own patients, except in cases involving joints, has the bone refused finally to unite."

I have myself never seen the union of an ordinary fracture prevented by age.

There was recently in Lawrence ward a female, æt. 75, who repaired, very rapidly, a compound and comminuted fracture of both bones of the leg; and fractures at the neck of the thigh bone in old people are usually mended in the ordinary time, not indeed by bone, for the separation of the bone fragments and of the periosteum, or absence of the latter as in intracapsular fracture of the head of the femur, commonly prevents this, but very serviceably by fibrous tissue; indeed Mr. Charles Miles has given us the particulars of the case of a man, æt. 85, who died seven months after sustaining fracture of the neck of the right thigh bone, and of the radius and ulna of the corresponding side. The latter united perfectly in six weeks, and in the posterior part of the fracture of the femur firm bony union had also taken place.[1] Mr. Holden has recently had under his care, in Colston ward, a man in his eighty-ninth year, who repaired a fracture of the shaft of his left femur.

Even paralytic old people repair a fracture rapidly, as in the following cases:

[1] St. Bartholomew's Hospital Museum, C. 181.

James P—, æt. 65, suffered a paralytic attack of the right side, from which he had not recovered when he fell and broke his right thigh just below the trochanter major. He was extremely restless, and constantly displaced the apparatus, but the fracture was firmly united at the end of the fourth week.

James P—, æt. 68, was admitted into Kenton Ward, in October, 1853, with a fracture of the right femur, having for some years been hemiplegic on the same side. This patient recovered rapidly, and was discharged, with shortening to the extent of half an inch, on the forty-sixth day after the accident.

At the opposite time of life, children who are paralysed will be found to unite a fracture readily. During January, 1855, William W—, æt. 9, was under treatment at St. Bartholomew's, having slipped and fallen heavily on his left side. This side was paralysed, and wasted from his early youth, and, owing probably to the helplessness of the muscles, there was no shortening, and scarcely any displacement of the left thigh, where the femur was broken across about its middle. Union was perfect on the thirty-sixth day. It is needless to repeat examples of similar results.

Apart from personal experience, I should mention that certain cases have come to my knowledge in which skilful surgeons have found it impossible to obtain union of a broken bone in children of tender years. My friend and colleague, Mr. Thomas Smith, kindly informs me, however, that no such case has occurred in the practice of the Hospital for Sick Children in Ormond Street, and the only instance I can at all refer to from St. Bartholomew's is that of a child whose femur was bent, or broken, after the patient had been discharged from the hospital as cured.

But Mr. Coote has been so good as to give me the following note of a case of broken leg in a boy, æt. 5. The child was under treatment for rachitis, and whilst the mother was re-adjusting some simple orthopædic apparatus, she felt the bones snap asunder. The limb was wasted, and the bones were light and atrophied.

Despite treatment the fracture refused finally to unite, and after a last attempt to procure union by introducing ivory pegs, the limb was amputated, rather more than two years after the occurrence of the accident. Mr. Coote has also informed me of the case of a boy, æt. 8, who had a fracture of the middle of the clavicle which failed ultimately to unite.

I have heard of somewhat similar cases, and they seem to have this in common; when a broken bone fails to unite in a child, within the usual time, the efforts on the part of the surgeon to procure subsequent repair are usually unavailing; a fact which should make us careful in the treatment of these cases in their early stages, and should render us cautious of transferring them to the care of inexperienced attendants before union is firmly completed.

Pregnancy and the period of lactation have been supposed by some surgeons to interfere with recovery from a fracture. I have never noticed any such consequence of these conditions. The following case is to the point:

Anne Thorne, æt. 22, pregnant eight months, was taken into Harley ward with a fracture of the left femur just above the knee, which had been caused by a fall on the pavement. The knee-joint was fixed, and the thigh disfigured by sinuses, the results of disease in early childhood.

On January 13th, 1853, twenty-two days after the accident, she was delivered of a boy. On February 9th the limb was put up in gum and chalk, the femur having firmly united, and she was discharged well, fifty-six days after her admission.

The repair, after fracture, of a bed-ridden bone, shows the ability of nature to deal with these hurts under apparently unfavorable circumstances. A man, æt. 56, had been bed-ridden for some years before he sustained, at intervals of time, a fracture of either femur whilst he was being turned in bed. Both fractures are in our museum.[1] One was through the shaft, about two inches below the lesser tro-

[1] St. Bartholomew's Hospital Museum, series iii, 92, and C. 116.

chanter; the posterior portions united firmly and smoothly, but so that they form an acute angle. The left femur, broken at the same part of the shaft, united at an angle of 75°. A second fracture occurred through the same shaft a few days before death. The texture of the bones is soft, light, and spongy, and infiltrated with fatty matter.

It has been said that patients tainted with syphilis occasionally fail to unite a broken bone. A great number so afflicted are under treatment in our wards, but I never heard of any such showing delay in mending a fracture. There is, however, a case reported by Mr. William Taylor[1] of fracture of a humerus in a female, æt. 23, caused by external violence six years before death, while she was affected with syphilis. On examining the humerus, which is in the museum of the hospital, it is clear, although the fractured ends are united only by soft cellular substance, that abundance of new bone is formed upon the outer surface of the shaft immediately above and below the fracture, but the extremity of the lower portion of the bone has perished, and is in progress of exfoliation. Thus, whilst the death of this part of the bone, in no way attributable to the syphilis, was in itself enough to have marred the union of the shaft, this specimen shows that the constitutional taint had no influence over the production of bone, which was abundantly deposited about the broken mass.

Mr. Paget has been so good as to give me the notes of the following case, in which a fracture was rapidly repaired in a patient suffering from syphilitic disease.

"A gentleman, about thirty-five years old, had lived a rough life, and had syphilis in Australia. On his return to England he was thin and feeble, but had no evidence of syphilis, or any other disease, except pains in his limbs, and especially pain about the middle of his left arm. The pain was not sufficient to prevent his using the arm, and he led a fairly active life. In trying to pull down a cab-window with his raised left arm the humerus broke across its middle.

[1] St. Bartholomew's Hospital Museum, series iii, 58.

The fracture was treated in some ordinary way: three grains of iodide of potassium were given three times a day, and in six or seven weeks the fracture was well united. The union remained firm many months afterwards, when I last saw the patient."[1]

Perhaps as good proof as any that constitutional causes are wrongly held to account for non-union of a fracture is found in the results of the treatment of the poor patients in St. Bartholomew's Hospital, who are drawn from the classes in which such influences exist to a large and varied extent.[2] During the last seven years there have been 2376 fractures under observation in the wards, exclusive, that is to say, of fractures of the upper extremities. With the exception of certain fractures of the patella, and of the neck of the thigh bone, which commonly unite, if at all, by fibrous tissue, there has been but one case of non-union.

Delayed union, it is true, is occasionally met with, and re-fracture of a bone sometimes retards the recovery. In other words, some patients take a long time to mend a hurt, some are unlucky. That the process of repair should be tedious in some cases is no more than we might expect from observation of the manner in which other hurts, or even diseases, are recovered from; for instance, in the varying time required to repair mere surface ulcers; and there can be no objection to refer delayed union to varying constitutional conditions, but this is widely different from the assumption that a certain state of the constitution can absolutely prevent a broken bone from ever uniting.[3]

[1] Dr. Drage has favoured me with the notes of a case of compound fracture of the left leg complicated with fracture of the right thigh. These injuries were sustained by a male who at the time was under treatment for secondary syphilis. The broken bones were united in about eighteen weeks.

[2] I may add that erysipelas and, amongst children, scarlet fever and various infantile complaints have been noticed in our wards as complicating, but in no way retarding, the repair of fractures.

[3] Amongst our hospital patients 157 days, 195 days, and even longer periods have occasionally elapsed before union of a broken thigh has been secured.

Amongst various instances of re-fracture in adults I never saw any bad consequences result. One of our hospital patients had, however, to remain with us from July until the following February, from his stamping on the floor to test the strength of the repaired fracture, when the femur at once snapped asunder.

On March 16, 1865, a labourer, æt. 52, was placed under treatment for a broken thigh, which united in 160 days. Shortly after his discharge he fell and broke the same bone at the same place as before, and the repair was again very tedious. He was kept at rest in bed, with immoveable apparatus, and recovered, with firm union, in 122 days.

The single case of non-union of a broken thigh was under my own care whilst I was on duty at the hospital during August, 1864.

Having broken his left femur at the junction of its upper and middle thirds, a hawker, aged fifty-four, was under treatment for 280 days, when he was discharged, but the bone was not united. We did a great deal too much for this man. From the first it was difficult to get the thigh into good position. The ends of the femur bulged outwards at the fracture, and the foot was constantly everted. To remedy this the apparatus was frequently shifted and modified, and I have always considered that our well-meant handling of the broken bone was the cause of its failing to unite—we did not give it sufficient rest. This fracture was at first treated by extension, with a weight drawing from the foot, and various attempts were subsequently made to obtain union of the bone, but they were ineffectual, and the patient eventually limped painfully, with his limb encased in a supporting apparatus.

Abscess in the shaft, or necrosis, or, as in Sir James Earle's case, the interposition of muscle, may now and then account for the non-union of a broken bone, but these cases must be of rare occurrence. Some of these fractures, too, belong to a class altogether distinct from ordinary cases. I refer to the fracture of a shaft from mollities or atrophy,

instances of which have been described by Mr. Stanley and others; or from cancer; or from necrosis with loss of that periosteum from which new bone is usually formed; the loss of this tissue is also the cause of non-union in a shaft from which any considerable portion has been removed, so that an interval is left between the fragments, as in the fractures referred to by Sir Astley Cooper, where there was loss of substance of the tibia, whilst the fibula retained its length.

I am indebted to Mr. Chippendale for notes of the case of a man, aged fifty, of cachectic appearance, who was admitted into Rahere ward, under the late Sir William Lawrence, during October, 1856. About five weeks before, whilst exercising a pair of horses, the one he was leading started away from the other, on which he was mounted, giving his arm a severe wrench. An abscess formed the following week, and this, when opened, was found to lead down to bare bone. On the morning of his admission, whilst merely turning over in his bed, he distinctly heard and felt the bone snap asunder. When subsequently examined there was deformity, with shortening and preternatural mobility, and a considerable surface of the shaft was felt to be bare. Although his general health improved, yet, despite treatment, the humerus showed no tendency to unite, and at last, the patient having been previously weakened by bleeding from a wound made to let out some deep-seated pus, it was found necessary, forty-four days after the accident, to amputate the limb. The man's condition became rapidly worse, and he died ten days later.

On examining the amputated portion a large sequestrum of bone, about four inches long, was seen to be almost detached from the shaft above, and quite so below where the fracture had occurred; no new bone had formed around the dead portion.

Mr. Charles L. Parker has presented to our museum[1] a tibia from which a portion of the shaft, nearly four inches in length, and including both the walls and the medullary tube,

[1] St. Bartholomew's Hospital Museum, series i, 262.

separated after necrosis. The remaining portions of the shaft are connected by a thick band of tough ligamentous tissue, attached to their gradually attenuated ends. They are also soft, light, and smooth on their surface, as if extremely atrophied. The fibula is healthy.

These parts were removed from a girl in whom the disease had long existed, and who had been quite unable to use the leg in walking.

The following case is described in the third volume of the catalogue of our museum.[1] The patient, a strong muscular man, suffered from a hard cancer of the breast, first noticed twelve months before his death. In the last two months of his life both humeri were fractured by slight forces. "Large portions of the medullary cavity of one of the broken bones are filled with a compact, very firm, greyish substance, like that of the common forms of scirrhous cancer," the bone tissue having completely disappeared before it. The walls are also thinned and in parts destroyed by the cancer, which even protruded through them. No union had taken place.

It is evident, I repeat, that these and similar cases are governed by conditions quite unlike those which surround ordinary fractures, and should not be mixed up with the latter in the consideration of the causes of non-union of bone.

From the preceding observations I conclude that some commonly assumed causes of non-union (as distinguished from delayed repair) of fractures may be fairly criticised. In writing of fractures which fail to unite, it is clear that three well-defined varieties must be enumerated.

(1.) Fractures, not inaptly termed spontaneous, which issue from diseases of bone, such as abscess, necrosis with absence of new bone formation, or cancer supplanting the osseous tissue, or such a condition as exists in mollities ossium. With such, it is evident that no union is likely to take place, for the bone is not merely broken, but its very nature is perverted or destroyed at the site of the fracture.

[1] St. Bartholomew's Hospital Museum, series i, 290.

(2.) Fractures (*a*) with separation of the bone and periosteum to such an extent that there cannot be thrown out bone material enough to fill up the gaps between the fragments; (*b*) occurring through bones not provided with periosteum (as in intracapsular fracture of the femur), and where it is difficult to keep the broken ends together.

(3.) All fractures other than the preceding; and these supply the large number of cases of so-called non-uniting fractures requiring treatment.[1]

It is with reference to this third class of cases that constitutional causes of non-union are so systematically enumerated,[2] but such causes are illustrated by evidence of a contradictory character, and we have, I believe, reason for ignoring the unfavorable influence of age, of paralysis, of pregnancy, of fevers. Rather than attribute non-union of fracture to influences of at least so doubtful a nature, it seems to me desirable to admit the force of Mr. Amesbury's statement, respecting the bad management of a broken bone, such as is involved in tight bandaging, insufficient rest, and so forth; or to accept the proposition of Malgaigne, that "when union is deficient, it is generally the treatment that is in fault." Without denying the possible occurrence of non-union of bone from causes beyond the control of the surgeon (apart from the local obstacles I have referred to), we find nature so apt at mending a fracture under the least favorable circumstances, and she so very rarely fails that, when she does, it is, I consider, always right and necessary to criticise treatment before finding fault with the constitution.

I have elsewhere referred to the coagulation of blood in veins, in consequence of bruising of the soft parts about a fracture, and to the extension of the clots from the lesser to the greater vessels, until they may come to obstruct the

[1] Nine cases of ununited fracture have been admitted into St. Bartholomew's during the last three years.

[2] These cases may be read in the 'System of Surgery,' article "Fractures;" in Malgaigne's work; or in Hamilton's 'Treatise on Fractures and Dislocations.'

chief vein of a limb. I have also mentioned several cases in which this condition was met with at after death examinations. Some few weeks ago, an old man died in the hospital, after mending a fracture in the upper part of the thigh, but the circumflex veins and the profunda were still filled with firm but partially decolorised coagula, and the venous circulation of the limb had been imperfectly carried on. The œdema which is associated with this condition is very characteristic, and I have thus referred to it: "This œdema often embarrasses the repair of fractures, more especially when the breakage is near the medullary foramen, or involves the laceration of a large vein, or when the tissues around are greatly bruised from the direct violence of the injury, so that clots form in the principal vessels. As this complication is of such common occurrence, it is strange that attention has been so little directed to it in cases of tardy repair or non-union. It may be suspected when, about the fourth week after the injury, the parts continue or become œdematous, cold and often aching. . . . It is always a serious affection, as shown by the frequency with which opportunities offer for inspecting the bodies of old and feeble people, who die some weeks after a fracture with no trace of union, but with numerous ecchymoses and plugged veins around the seat of injury."[1] In cases such as these the whole of the venous blood is commonly thrown into the superficial veins, and it becomes important in every way to facilitate the circulation through them. Under such circumstances, it is needless to insist upon the damage occasioned by tight bandaging, or to show how materially it may hinder the return of blood from the limb. So far as my experience goes, fractures of the forearm are most liable to suffer from this cause, from the facility with which pressure can be applied to the superficial vessels between the wrist and the elbow.

It is impossible to exaggerate the hurt sustained by a joint, owing to the non-union of the long bone which serves

[1] 'System of Surgery,' vol. iii, p. 300.

as a fulcrum for the efficient performance of its chief movements.

During the year 1854, there was under the care of the late Mr. Stanley, in Darker ward, a man, æt. 27, who had an ununited fracture of the femur. The hurt had been treated elsewhere, thirteen months before he came to the hospital. Ivory pegs were inserted into holes drilled through the broken ends of the bone (which were also wired together). In the end no good resulted from the irritation set up by the pegs, and the limb being painful and useless the patient begged to have it removed. Amputation was eventually performed at the upper third of the thigh. Severe recurrent bleeding caused great exhaustion, but the man eventually recovered.[1]

The effect of non-union upon the movements at the knee was well marked in a case for permission to use which I am indebted to Mr. Paget, and which is also of interest because of the nature of the disease which prevented the broken bone from uniting.

A man, æt. 27, was admitted into St. Bartholomew's in January, 1864. He had long roughed it in New Zealand, and had complained for two years of intense pain in the lower part of his right thigh; but thirteen months ago, as his knee began to swell, this pain by degrees disappeared. The limb, however, presently became useless, and the joint could, as it seemed, be twisted in almost any direction, as if from great laxity of its ligaments, flexion being limited by the great thickening about the end of the femur. Coarse rubbing and crackling were felt when the joint was moved, as though portions of thick fibrous tissue, or even bony materials, jostled one against another.

The limb was useless, the knee movements were lost; splints were quite inefficient to give support or relief, and his age justified, as the necessities of his life needed, the removal of the limb. After amputation, the lower end of the femur was found to be expanded into a thin-walled cavity, and filled with what looked like dried-up pus. Just

[1] Part of the bone showing the non-union is in St. Bartholomew's Hospital Museum, C. 121.

below the situation of the line of the epiphysis, that is, through the condyles, the bone was broken across, and the broken surfaces had become smooth and hard. This fracture had allowed the free movements which had been so deceptive, for the joint itself with its ligaments was free from disease. The patient made a slow recovery, in the course of which it was necessary to tie the right common femoral artery for severe secondary bleeding from the stump.

In none of the cases of long standing non-union of the femur which I have seen has any practical good been derived from the use of the apparatus, which has been devised to supply the stability lost by the lack of continuity in the bone, with the object of enabling patients to get about. I join with those who are of opinion that for this condition amputation may be ultimately necessary, if the patient is not advanced in years, as the limb is useless for working purposes, a constant trouble and a source of pain; always premising, however, that amputation should never be thought of until every effort has been made to get the bone to unite, for indeed it is surprising how very long a time after the occurrence of the injury union may take place.

To this end the treatment most likely to be successful, when the femur is the bone concerned, is that which ensures rest in bed with the limb secured in good position, but without any circular compression, for two or three months, or longer if necessary; and I believe that the good results which are said to follow certain remedies, and various operations for the cure of these cases, have issued from the quiet and immobility which at the same time have been rendered necessary. It is easy to believe that remedies which improve the general health are advantageous, but it is less easy to understand the recommendation that a person suffering from non-union of a broken thigh should be up and about and using the limb, which it is said should be secured in some fixed apparatus. It is true the health may be bettered by the exercise and the change, but it is very questionable whether the process of repair is expedited. So far as I have

had opportunities for noticing these and other cases of only slightly delayed union, they should certainly be kept in the horizontal position until the bone is fixed, and should not be exposed to the risks, and at best the uncertain good, of the patient's moving about, for no arrangement of apparatus can then be relied on to ensure immobility of the femur, and without security for this no repair can be confidently looked for.[1]

With reference to the great length of time after the lapse of which a fracture, if properly treated, may be repaired, and as indicating the treatment which may be followed, I add to those previously referred to the following histories.

In the spring of 1865 a soldier, recently discharged from a cavalry regiment, æt. 23, and in good general health, came under my care with ununited fracture of the right humerus at the upper part of its lower third. The arm had been broken whilst he was in India, twenty-seven months previously, by a fall from his horse. It was now useless, for he was unable either to flex or to extend the forearm, because the attempt to do so doubled up the humerus at the seat of its fracture.

Some time after his admission into St. Bartholomew's I cut down upon the bone, following a line in front of the course of the musculo-spiral nerve, which was seen during the operation lying somewhat in the way, and exposed the broken bone ends. These tapered to a point and were loosely connected by tough fibrous tissue, indeed they tapered so gradually that a considerable portion had to be removed from either before any breadth of surface could be obtained. The raw bones were then wired together, and the parts were fixed in an angular tray and were kept at rest for two months. The wire was then removed, but no union having been established the arm was again fixed and kept at rest for two months longer. At the end of this time union was not

[1] These remarks do not apply to fractures of the leg, which can be easily and securely 'put up' in plaster of Paris, or in gum-and-chalk bandages, and the patient can be allowed to get about without incurring any risk.

absolutely firm, but, with slight external support, he was able to leave with a useful arm and elbow-joint.

I think it has escaped general notice that a broken humerus may be very serviceably united by fibrous tissue if time is allowed, before the arm is brought into use, for the thorough contraction and hardening, if the latter term may be permitted, of the material joining the two portions of the shaft.

To the description of a case of non-union by bone of the middle of the shaft of the humerus it is added by Mr. Brendon,[1] "the use of the arm was so little impaired by the fracture that the patient worked as a sailor to the time of his death." In September, 1855, a man, æt. 29, was in Darker ward with an ununited fracture of the humerus, just below the insertion of the deltoid, of nine months' standing. The arm was kept fixed in an immovable apparatus for four months, and the union, not osseous, was so far completed by the great thickening of the tissue about the fracture that the patient could use the limb nearly as well as its fellow.

I had recently an opportunity of examining the parts in a case in which fibrous tissue had united a fractured arm. In May, 1862, a sailor fell from the main topgallant yard, and in his fall struck his left thigh and arm across some spars stowed midships. The fractures which resulted were treated by the ship's carpenter. He was then eighteen. In July, 1864, he was admitted into St. Bartholomew's, under the care of Mr. Paget. The humerus was firmly united, but the two portions of the shaft were fixed at a right angle at the upper third of the bone. When this fracture was operated upon for the cure of the deformity it was found, contrary to expectation, that union was not osseous but by tough fibrous tissue.

With reference to the support given by parts around the fractured ends, there is a curious specimen in our museum[2]

[1] St. Bartholomew's Hospital Museum, series iii, 2.

[2] Ibid., series iii, 65.

in which the disjoined portions of a broken humerus are held together by a distinct ring of bone which completely surrounds them.

Such cases should encourage us to persevere with treatment for non-united fractures, more particularly of the humerus, even when but a small chance apparently remains of osseous repair, yet this may follow years even after the occurrence of the break.

I have before me the original notes of the case in which the late Mr. Stanley obtained bony union in an ununited fracture of the bones of the leg, after driving ivory pegs into the ends of the disjoined portions. The fracture, a compound one, had occurred two years previously. For twelve months the patient tried to get about with the help of an apparatus, and after this he came up to town. Blistering was then employed for two months, when he was discharged, but returned the following January, and two years and four months after the occurrence of the accident the ivory pegs were introduced, and four months later the bones had firmly united. In this case, as in one previously referred to, the pain caused by movements of the bones, as in the simple act of crossing one leg over the other, was remarkable, and was the constant complaint of the patient.

An equally good result was obtained by different means in a case under Mr. Paget's care, during the years 1866-7. The patient, a Deal boatman, æt. 35, a robust-looking man, had a broken humerus of twelve months' standing. The fracture was in the upper part of the lower third of the shaft. Soon after his admission, the bone was fixed by a metallic piercer after the plan recommended by Bickersteth and Gaillard,[1] but no good resulted from the operation. Rubbing the broken ends together, and confinement in a gum-and-chalk bandage, had no good effect, and eventually the limb was freed from all bandages and simply supported on an angular splint, which was swung during the daytime

1 'Med.-Chir. Trans.,' 1864, and 'New York Journal of Med.,' 1865.

from a 'davit.' The patient was thus enabled to take active exercise without the least risk of disturbing the ends of the bone. In three months the bone was united, and two years and two months after the occurrence of the fracture the union was firm.

There exists a very general fear for the ultimate condition of joints which become stiff from disease during the repair of a fracture. I do not refer to joints which are actually broken into, but to such as lie near to a broken shaft, as the elbow with fracture of the humerus, or the knee with fracture of the lower part of the femur. From this fear comes the recommendation, which may be read in most surgical works, but which is certainly a bad one, to make passive movements of the joint at an early date, to guard against the possibility of stiffness; "hence," Malgaigne writes, "the surgeon as soon as the apparatus is taken off should move the joints, forcing those which are stiff, and advise the patient to continually exercise them in this way. Next day and the day following he should keep it up, until the articulations have recovered their full freedom."[1]

Dr. Hamilton has described the bad effect on a fractured humerus, produced, he believes not infrequently, by the weight of the forearm, or of apparatus continued beyond the elbow when used for a broken arm, dragging upon the lower portion of the shaft and displacing it from the upper; he considers that this, with the attendant and frequent shifting of the lower end of the bone, is a constant cause of delayed union, and even of non-union, of the humerus. At St. Bartholomew's Hospital, fracture of the arm above the condyles is never encumbered with heavy apparatus. The shaft is kept in position by three or four light and well-padded wooden splints, a short one reaching from the inner condyles to the armpit, and others from the elbow to above the shoulder; these are held together by straps, which can be readily tightened from time to time, and the forearm is slung with bearings from the wrist. The great advantage of this plan of

[1] Translated by Packard, p. 241.

treatment consists in the entire freedom of the elbow, which, far from becoming rigid, as in the cases observed by Dr. Hamilton, allows of chance movements of the forearm being made without risk, because they hinge entirely upon the joint, as is natural to them, so that the fracture is not disturbed.

When, however, from the necessity of the case, or from the treatment employed, the elbow has become stiff, there is every reason to believe, as Mr. Paget suggested to me some years ago, that non-union of the humerus is risked by the well-meant attempts which are made by the surgeon, or by the patient, to move the joint before the new bone is thoroughly firm; and how likely it is that the fracture will recur from such attempts is evident when the powerful leverage used in making passive movements at the elbow is borne in mind.

This risk alone, and the same holds good for other articulations, should make us careful not to be in any hurry to remedy the rigidity which follows from temporary disuse of a joint; it is certainly not worth incurring when it is known that this stiffness is all but invariably recovered from in time.

There was recently under my care a labourer, æt. 38, who fell and fractured the upper third of his left tibia. The bone united as usual, but the knee remained stiff, the movements of the joint were slowly recovered in four months. In a case of compound fracture in the lower third of the leg, nearly a year passed before the ankle-joint movements were regained. And perhaps it would be well if it were made more clear to a patient than it often is, that although a fracture will unite in a given number of weeks, yet that complete recovery from the injury is always an affair of months, and when joint stiffness is superadded of six months, or even more.

For all joint stiffness resulting from disuse during the repair of a fracture, passive or other movements should never be attempted until the broken bone is firmly consolidated, and then only with great caution.

In the preceding notes I have not thought it necessary,

for obvious reasons, to refer, in individual cases, to the meddling with recent fractures which still seems to be, unfortunately, of common enough occurrence. I have been obliged to hear from patients that the splints, or other supports, have been removed daily, or every few days, to see how the repair was going on. Nothing could be more reprehensible. As a rule, a fracture is badly "put up" which requires any subsequent moving or disturbing of the apparatus.

From what I have observed respecting non-uniting and slowly uniting fractures, I arrive at the following conclusions:

Non-union of an ordinary fracture should never occur.

Under careful treatment bones will unite two years or longer after the occurrence of the fracture.

It is reasonable to suppose that such fractures would have united at an earlier period if properly treated.

Treatment of delayed union (slowly uniting fractures) should consist—

First, in the improvement of the health.

Secondly, in placing the broken bone in the best position attainable, and in the avoidance of local obstruction to the circulation.

Thirdly, in leaving it at rest until it unites; its doing so being simply a question of time.

Fourthly, in avoiding all attempts to overcome the stiffness of joints adjacent to the fracture, until the bone is firmly united; and this applies also to the management of fractures which unite in the usual time.

A

CASE OF AXILLARY ANEURISM ON THE RIGHT SIDE,

FOR WHICH THE

SUBCLAVIAN ARTERY WAS SUCCESSFULLY TIED;

THE LEFT SUBCLAVIAN ARTERY HAVING BEEN LIGATURED IN THE SAME SUBJECT FOR AN AXILLARY ANEURISM ABOUT THREE YEARS PREVIOUSLY.

BY

EDMUND J. FURNER, F.R.C.S.,

SURGEON TO THE SUSSEX COUNTY HOSPITAL.

COMMUNICATED BY

SIR WILLIAM FERGUSSON, BART.

Received Dec. 23rd, 1867.—Read April 14th, 1868.

J. P—, æt. 30, was admitted into the Sussex County Hospital under the care of Mr. Furner, October 22nd 1866.

He was a well-made healthy-looking man, with a florid complexion, and had enjoyed good health. He had been employed on the Brighton Railway, his occupation being very laborious, obliging him at times to exert all his strength in lifting heavy weights, such as the carriage wheels, &c. He had occasionally drank freely, principally beer or porter.

In June, 1863, he was first under the care of Mr. Furner, in consequence of an aneurism of the left axillary artery, for which the subclavian was ligatured, and he was discharged cured in about eleven weeks.

The case was published in the 'Medical Times and Gazette' of October 24th, 1863.

For the first twelve months after leaving the hospital he was employed in lighter work, but ultimately resumed his more laborious occupation.

About two months prior to his readmission, whilst lifting a heavy weight, he felt a sharp crack in the right shoulder, and the arm seemed numbed for some minutes, but he was able to return to his work in a quarter of an hour; five days afterwards he observed a swelling about the size of a walnut below the right clavicle; this at first was not attended with much pain, and did not prevent him following his occupation until three days before his admission into the hospital, when the swelling had rapidly increased in size, and then measured seven inches in length by five inches in breadth.

The tumour was situated beneath the clavicle, pushing up the bone; it extended outwards into the axilla, and inwards towards the sternum. It was well defined, firm and elastic, having a loud thrill which could be felt and heard; upon pressing the subclavian artery the pulsation was readily controlled; a loud murmur was audible over the whole of the tumour.

There was considerable pain, but no œdema of the arm. The pulse could be easily felt at the wrist.

He was ordered to lie in bed, and cold evaporating lotion applied to the tumour; to have moderate diet, and an aperient. During the night of the 23rd there was great pain in the tumour, chiefly in the neighbourhood of the axilla; it had considerably increased in size, and the arm was slightly œdematous. Mr. Furner saw the patient about 2 p.m. of the 24th, and determined to operate without delay. The usual incision was made in the line of the clavicle; some of the fibres of the clavicular portion of the sterno-

mastoid muscle were divided, and several small vessels required to be tied; the external jugular vein was secured with a double ligature, and divided. Great difficulty was experienced in consequence of the tumour pushing up the clavicle, and thus causing the vessels to lie at a considerable depth. A large artery in the situation of the subclavian was brought into view, and a ligature passed around it, but not tightened, as pressure upon it with the finger did not control the pulsation in the tumour; this vessel was held aside, and the subclavian reached at a great depth, and ligatured, when all pulsation in the tumour ceased.

In close contact with the ligature was a large artery, most probably the transversalis colli having an abnormal origin, arising from the subclavian.[1] A ligature was placed upon this vessel also. The pleura which was seen at the inner angle of the wound moving with each inspiration was not injured.

The operation lasted upwards of an hour, and was borne by the patient with great fortitude. Chloroform was not given. The arm was wrapped in cotton wool, and the patient removed to bed; shortly afterwards he had severe pain in the arm, extending down into the hand, which was relieved by change of position.

October 25th.—He had slept at intervals during the night, and had taken beef-tea and milk freely; he was tranquil, complaining only of a pricking sensation in the wound, which looked healthy, and discharged a little serum tinged with blood. The skin was cool and moist; pulse, as felt in the temporal artery, 72. Tongue clean. He had passed urine naturally, and it was healthy in character. Without detailing the daily symptoms, it is sufficient to say that he progressed most favorably, requiring no medicine, except an occasional aperient. The diet consisted of beef-tea, arrow-root, milk, eggs, &c. He slept many hours daily, without a sedative; his skin acting then most profusely. The tumour gradually, but perceptibly, became smaller.

[1] This was also the case in the former operation. See 'Medical Times and Gazette,' October 24th, 1863.

November 1st.—Six ligatures came away in the dressing from the smaller vessels.

4th.—This being the twelfth day from the operation, his diet was slightly reduced, in order that the vessels might not be overcharged. There was no unfavorable symptom of any kind. The tumour had much diminished in size, was soft and elastic, but no pulsation could be detected.

7th.—The ligature from the transversalis colli artery came away fourteen days after the operation.

14th.—The granulations being a little flabby, he was allowed a glass of sherry daily, and his diet was increased.

17th.—The ligature from the subclavian came away twenty-four days from the operation, and he continued progressively to improve.

December 20th.—The wound had almost healed, and his general health was good. His arm being bandaged to the side he was allowed to get up, and to go out of doors when the weather permitted. He continued well up to January 10th, twelve weeks after the operation, when he complained of pain over the sternum. The tumour now began to increase in size, accompanied by pain and fever, with an irritable cough. Leeches were applied without much benefit. The tumour continued to increase in size, and at length, when an obscure sense of fluctuation was experienced, a free opening was made, and about eighteen ounces of most offensive matter, with broken-up coagula, were discharged. After this he made a rapid recovery, and has remained in good health since. When presented to the Fellows of this Society eighteen months after the operation, he was in robust health, and had resumed his former laborious occupation.

CASE

OF

CYSTIC DISEASE OF THE KIDNEY

SIMULATING OVARIAN DISEASE.

BY

HENRY COOPER ROSE, M.D., F.G.S., F.L.S.

Received Feb. 27th.—Read May 12th, 1868.

At a period when the subject of ovariotomy is engaging the attention of surgeons in all parts of Europe, every case which tends to throw light upon the matter, either in rendering the diagnosis clear, or in pointing out difficulties to be overcome, cannot fail to be of interest. I do not hesitate, therefore, to draw attention to the following history as one capable of conveying some useful lessons.

C. R—, a somewhat delicately constituted young lady, æt. 18, came under my professional care in January, 1856. Menstruation had commenced at the age of thirteen, but had never been regular, there being intervals of three or four months in the catamenial periods. Two years before (in 1854) she had observed blood in her urine, but had never spoken of it. This symptom continued occasionally for

some years. She stated that a swelling had existed low down in the left side in the inguinal region for some time.

On examination I found a tumour occupying the position of the left ovary about the size of a large orange. In the following May there was tenderness over the left side, extending from the edge of the false ribs to the inguinal region, with a distinct prominence. The uterus was moveable and normal in size. The tumour which filled the space appeared to be solid—no fluctuation was perceptible. Her general health had not suffered much. In the September following, the tumour was larger, and there was a sense of deep fluctuation, but this was questionable. Hæmaturia had been more frequent, and sometimes pain of a dull, aching, character was complained of. Gallic acid always relieved the hæmaturia; and she had continued to menstruate as before, every two or three months.

From this time until November, 1858 (a period of two years), she had been much better, the tumour remaining very much in the same state. Now, however, she had a severe attack of hæmaturia, which weakened her considerably.

The tumour now occupied the whole region from the diaphragm to the pubes and Poupart's ligament of the left side. It was very prominent a little to the left of the umbilicus, and it extended beyond the median line into the right side of the abdomen. Fluctuation was very perceptible. Tenderness was excessive over the whole surface of the abdomen. It was found on examination that the tumour occupied but a small portion of the pelvis.

A few leeches and fomentations relieved the pain and tenderness; and until December, 1861, a period of *three more years,* very little change took place in either the tumour or her symptoms, except that she felt much better, although purulent matter had been found in the urine. She had passed some weeks at the sea-side in the summer, returning stronger and better in all respects. Several medical friends had seen her, who were all of opinion that it was a case of ovarian disease associated with hæmaturia.

In June, 1861, Mr. Spencer Wells saw her, and

expressed the opinion that an ovarian cyst, or a pelvic abscess, communicated with the bladder, but discouraged a proposal to make any freer communication with the bladder or to inject iodine. The tumour remained almost unchanged in size or position until June, 1865, a period of *four more years*. At that time it began rapidly to increase, and on July 22nd, 1865, I was hastily summoned into Gloucestershire, where the patient was on a visit. There I met Mr. Child and Mr. H. Cooper; and finding the whole abdomen very tense and painful, and a prominent point situate equidistant between the crest of the ilium and the umbilicus on the left side, which was livid and the sac thin, I introduced a trocar and evacuated seven pints of sanguineous clotty fluid. The sac was very tough. A large mass remained in the abdomen, which appeared to be partly solid and partly multilocular. She continued very faint and sick for many hours after the operation, but all pain was relieved; and by degrees she so far recovered her strength as to be able, *two years afterwards*, to take moderate exercise, and even to play croquet for three or four hours at a time.

The opening made remained patent, giving vent to fetid discharge, which varied much in character and quantity from time to time, sometimes soaking through her clothes, at other times being very small in amount.

A year after the operation Mr. Spencer Wells saw her again. At that time the fistulous opening was situate close under the ribs, taking a downward course. The tumour was reduced to the size of a man's fist. The os uteri could not be felt, being dragged upwards by the tumour. Some of the fluid from the tumour Mr. Wells examined, but none of the elements of urine could be detected.

In June, 1867, some anasarca of the legs took place, and albumen was found in her urine. This symptom gradually increased, and in September, 1867, vomiting became very troublesome, and she very *gradually* sank on October 29th, retaining consciousness to the end.

Mr. Gay made a post-mortem examination on the following day, and gave me the accompanying notes:

"Externally there was great fulness in the left lumbar region and the adjoining umbilical region *in front*. On percussing this space it was found to be dull as well in front as behind, whilst in the other regions of the abdomen percussion elicited a hollow sound. It was clear from a little manipulation that these physical signs were due to the presence below the integuments, which were moveable over it, of a large unyielding and somewhat irregularly oval-shaped body. There was a fistulous opening in the left hypochondrium, through which a probe was passed backwards and downwards in the direction of this tumour, prior to the abdomen being opened. On throwing back the integuments, this fistulous passage, the walls of which were very thick, was observed to lead to the supra-renal capsule, which together with the kidney formed the hard body alluded to. Together they formed a mass nearly six inches in its long, and above three inches in its short, diameter. The descending colon was closely adherent to it, but it had no other attachments in front, with the exception of that to the skin by means of the wall of the fistulous track just described. The mass was firmly bound down to the parts with which it was in contact *behind*, by close adhesion to the fascia over the atrophied quadratus lumborum muscle, and laterally by tough and thick fibrous bands which could only be severed by the scalpel. The surface of the mass was somewhat uneven, mottled, of a dark bluish and yellow colour, and still partially invested with fat. The kidney which was closely adherent to the supra-renal capsule formed a large cyst, from which, on cutting it through, a considerable quantity of thin ochry matter and shreds of bloody pus clot flowed. The tissues had become completely degenerated, the degeneration in all probability having begun with the membrane lining its pelvis and calyces. This membrane had become a thick, almost pulpy, dark greenish-coloured lamina, which appeared in the course of separating from its connections fragmentally; the tubular tissue had entirely disappeared, whilst the cortical was reduced to a very pale, thin stratum surrounded by the capsule, which in its turn had become very thick and almost

of cartilaginous hardness and appearance. *The ureter had degenerated to a hard cord and was entirely closed up.* The emulgent artery and vein were large. The supra-renal capsule had been hollowed out by a large sinuous passage which formed a continuous channel from the fistulous track to the kidney. Its walls were dense and evidently lined with mucous membrane. Between them and the external tunic of the capsule there were here and there traces of the normal tissues. The adhesion of the capsule to the kidney was so firm that it could not be torn asunder. The right kidney was enlarged to twice its normal size, and was undergoing yellow degeneration.

The ovaries, lungs, liver, and spleen, were healthy."

Remarks.—It is worthy of observation that this tumour had existed for *fifteen years,* that it had commenced and was first observed in the situation of the left ovary, that no pain was ever referred to the region of the kidney, that the urine was normal except upon those occasions when she suffered from hæmaturia, and that the uterus was latterly drawn up so as to render it difficult or impossible to reach the os with the finger; that in the early stages of the disease the uterus was moveable and apparently normal in size, and that when some of the fluid from the tumour was examined no elements of urine could be detected. Of course the post-mortem examination revealed the causes which produced these symptoms; and the total destruction of the secreting portion of the kidney accounts for the absence of urea in the abstracted fluid. With the exception of the hæmaturia, which naturally excited some doubt and question, there was nothing in the case which was not quite reconcileable with the diagnosis which had been formed of it by some of those most accustomed to the examination of this kind of cases. In fact, it became a question as to the propriety of removing the tumour should it become a source of much distress; and I well remember when I mentioned the case at a meeting of the Royal Medical and Chirurgical Society, after the reading of a paper by Dr. Lee on some cases of ovarian disease, that the fact of this complication of hæmaturia was not deemed

by some Fellows who replied as a symptom which should interdict ovariotomy even should it arise from direct communication with the tumour.

A similar case to the one I have narrated is published by Mr. Spencer Wells in the 'Dublin Quarterly Journal of Medical Science' of February, 1867, in which ovariotomy was attempted with, of course, a fatal result. The more frequently difficulties and dangers are pointed out, the greater will be the success in practice, and I therefore deem this instructive case not unworthy of record.

A HISTORY OF SEVEN CASES

IN WHICH A

MORBID GROWTH WAS REMOVED

FROM

ONE OF THE VOCAL CORDS

BY THE AID OF THE LARYNGOSCOPE.

BY

GEORGE JOHNSON, M.D. Lond.,

PHYSICIAN TO KING'S COLLEGE HOSPITAL.

Received March 25th.—Read May 26th, 1868.

Cases of morbid growths within the larynx are amongst those for whose exact diagnosis and successful treatment the use of the laryngoscope is essential. It will be seen from the narrative of the following cases which have come under my care during the last four years, that in more than one instance the patient had been subjected to a long and fruitless course of medicinal treatment before the true nature of the disease was ascertained by the use of the mirror.

The instrument which I have employed for the removal of growths from the larynx is a wire écraseur. Dr. Thomas J. Walker, of Peterborough, was the first in this country to remove a growth from the larynx by a wire loop (see 'Lancet,' November 9th, 1861). This instrument, which was made for him by Messrs. Weiss, consisted of "a

silver canula, much resembling Gooch's double canula, but longer, and curved at one end almost at right angles." A piece of thin iron wire was passed through the tube and returned so as to leave a loop, which thus formed a simple but very effective écraseur. With this instrument Dr. Walker removed from the larynx of a boy, aged fourteen, a polypoid growth which was threatening suffocation.

Messrs. Weiss subsequently made an improved écraseur of steel for Sir Duncan Gibb, which he has employed successfully in several cases.

The instrument which I have used is a modification of Sir Duncan Gibb's. Several minor alterations have been made, but the chief improvement which, at my request, was made by Messrs. Weiss, is that of a hook fixed on the upper surface, over which the forefinger of the operator passes. Thus, the forefinger antagonising the thumb passed through the ring at the end of the instrument, the operator has a firm hold of the écraseur, and with the aid of the mirror, which is held in the left hand, he can readily direct the wire loop towards the morbid growth.

The écraseur has this advantage over any form of laryngeal forceps, that, unlike them, it very rarely tears the mucous membrane or seizes any other object than the growth to be removed. It is a perfectly safe and harmless instrument.

The secret of success in these cases, consists in bringing the wire loop exactly opposite the wart at the time when the touch of the instrument excites the contraction of the glottis, by which the tumour is driven into the snare and then caught by the sudden retraction of the wire. I have always found that any portions of morbid growths that are torn off have been tightly held by the wire, which, partially cutting them through and embedding itself in their substance, thus brings them away. I have no reason to suppose that any particles have fallen into the trachea. If, however, this were to happen they would probably be expectorated as speedily as small masses of mucus are expelled.

Let me for a moment direct the attention of the Society to a simple and efficacious light condenser, which has been

made at my suggestion by Messrs. Meyer and Melzer, of Great Portland Street. It consists of a pair of iron spring forceps which may readily be fastened on to the chimney of almost any form of lamp. Into this, in front of the flame, is fixed a plano-convex lens, and, behind, a copper reflector silvered. The instrument can readily be taken to pieces, and the lens, with its holder, may be packed in a case of convenient size for the pocket.

The following narrative of cases is given in the order in which they occurred.

Case 1.—J. W—, æt. 49, a cabinet maker, was sent to me in March, 1864, by Dr. Tyler Smith. His voice was extremely husky and feeble; about twenty years ago he caught cold by sleeping in a damp bed: he became hoarse and has continued so ever since. For about ten years he was almost constantly under medical treatment; he had been repeatedly blistered and once salivated; he had taken much cod-liver oil, and he summed up his opinion of the effect of all this medication by saying that "it had injured his general health and had not improved his voice," which in fact had steadily become more feeble and husky. He had never suffered from dyspnœa, and there was no stridor during a forced inspiration. On examination I found a wart, nearly as large as a mulberry, attached to the anterior part of the right cord and occupying a large portion of the glottis. The symptoms were thus explained and the failure of former medical treatment was accounted for.

This was the first case of warty growth that had come under my own care. I was then comparatively unpractised in the application of instruments to the larynx, and I made many unsuccessful attempts to remove this growth, sometimes with the forceps, at other times with the écraseur. All my success was with the écraseur. On one occasion, during a sitting of about ten minutes, I succeeded in catching the wart three times out of five, and each time I removed a piece of considerable size. On another occasion I had removed a small portion with the wire, and after the patient left my

house he expectorated a small piece which probably had been loosened by the wire and then driven off and expelled during the effort of coughing. At length I succeeded in removing nearly the whole of the growth, but there still remains a nodular projection on each cord near its anterior end, and there is a general want of smoothness and polish over the surface of both cords. These appearances explain the fact that his voice, although stronger and clearer than it was, is still feeble and husky.

Case 2.—Mr. H—, æt. 50, engaged as a lay agent for the Pastoral Aid Society, consulted me first in October, 1863, on account of hoarseness, which was much increased by continuous speaking even for a few minutes. The feebleness of voice had existed for more than a year, and he accounted for it by his habit of addressing crowds in the open air. When I first examined his larynx I saw only an injected state of the mucous membrane, but after repeated examinations I plainly saw a red and apparently a very vascular tumour between the two cords just at their insertion. During inspiration this tumour disappeared below the glottis, but during vocalisation it was driven upwards between the cords and prevented their close apposition. The feebleness of voice was now explained. I repeatedly applied a solution of iodine on a bent brush, and, although I sometimes fancied that the tumour had somewhat diminished, I at length arrived at the conclusion that no good had been done by its application. I then determined to attempt the removal of the tumour by the écraseur; and on the 27th June, 1865, after an attempt to catch the tumour, there was an escape of blood from the throat; altogether about an ounce of blood was expectorated, and in the midst of it a shreddy piece of membrane, which I have preserved as a microscopic specimen. Now, on examination of the larynx, I found that the tumour had entirely disappeared, and in place of it there was only a slightly uneven projection near the anterior extremity of the right vocal cord, where the tumour had been attached. I infer that the tumour had been a vascular cyst containing fluid—

that the cyst had been ruptured by the grasp of the wire loop, and that the membrane which was expectorated had formed a portion of the cyst-wall. The voice, for a few days after the removal of the tumour, did not appear to be much improved, but gradually it became stronger and clearer. I occasionally touched the spot where the tumour had been attached with a solution of nitrate of silver; the nodular unevenness quite disappeared. He was soon able to resume his speaking and reading aloud, and when I last saw him, in December, 1866, he told me that his voice continued good.

Case 3.—J. G. C—, æt. 59, a manager of large estates in Ireland, consulted me in May, 1865, on account of hoarseness, which he said had come on rather suddenly without any symptoms of cold in October of the previous year. He was blistered without benefit. In the middle of December he consulted Dr. Smyly, of Dublin, who told him that he had a wart on his right vocal cord. In March, 1865, he saw Dr. Stokes and Dr. Smyly together. He came to me on the 19th May, 1865, and, without giving me his history, begged me to look at his throat and tell him the cause of his hoarseness. I at once saw a wart about the size of a pea, but of a somewhat conical form, on the right vocal cord near its posterior or arytænoid end.

I made many attempts to catch the wart with the écraseur, which were entirely unsuccessful, until I discovered that in consequence of the unusual length of the patient's neck, he being a very tall man, it was necessary to have the bend of the écraseur much deeper than usual. This being done, on the 18th June I pulled off a piece of considerable size, and the voice became clearer immediately. On the 20th June a smaller piece was removed with a yet further improvement of the voice. After this some granular particles came away on the wire, and the cord was left with only a slightly conical smooth projection where the wart was attached. When he left me on the 26th June, he said his voice was then as strong and clear as it had ever been. I saw this gentleman on the 5th July, 1867. I then observed a somewhat

granular thickening of that part of the cord where the wart had grown. His voice was, as he said, "not so much hoarse as gruff and reedy, and differing as much from what it formerly was as a note on a bassoon differs from the same note on an organ-pipe." He said that as his voice was sufficiently good for any use that he had to make of it, whether in reading or in speaking aloud, he did not care to submit to any further treatment. He had only called on his way through London, as I had requested him to do, to report progress.

Case 4.—J. L—, æt. 26, an assistant in the establishment of Messrs. Bell and Co., the chemists, in Oxford Street, consulted me first in the summer of 1864, on account of hoarseness, from which he had suffered for many years; in fact he could not remember that his voice had ever been clear and natural. He had seen many doctors, and had taken much medicine, including cod-liver oil, on account of a supposed tendency to phthisis, but his voice had remained husky and feeble. He had no cough, and there were no signs of disease within the chest, but, on examination with the laryngoscope, I at once saw a wart about the size of a small pea, near the anterior insertion of the right vocal cord. This wart explained the hoarseness. After several attempts I removed a small piece of the growth by the wire, and the voice was somewhat stronger. I made no further attempt to remove the growth until the following summer. In the mean time I had had considerable practice in manipulating the throat, and had improved the écraseur. At length, on the 23rd June, I caught the wart with the wire and removed it entire, leaving the vocal cord with only a slight unevenness on its surface, which disappeared in a short time. Immediately after the removal of the wart, the voice became strong and clear, so that he could sing as he had never been able to do before. The patient has since married, and taken a business at Darlington. I wrote lately to inquire about his voice. In his reply, which is dated May 18th, 1867, he says, "In answer to your inquiry, I am

happy to say that my voice remains clear and strong, and if I were a singer I could pitch either a high or a low note with perfect ease."

Case 5.—Mr. J. W—, æt. 68, a surgeon, was sent to me by his friend Mr. Barnard Holt, in February, 1867. He had been hoarse for nine months, and for the past six months his voice had been so feeble as seriously to interfere with the discharge of his professional duties, and he had been quite unable to go into society. On laryngoscopic examination I immediately saw a wart about the size of a pea, but of a somewhat conical form, on the right vocal cord, near its anterior extremity. During inspiration the wart fell below the glottis, but during vocalisation it was driven by the expiratory current between the cords, so as to keep them apart, and thus to render the voice very feeble and husky. He had a troublesome cough, which apparently was mainly caused by a very long and congested uvula. I advised him to have the end of the uvula cut off; this was done by Mr. Holt, and was attended with the double advantage of relieving his cough, and greatly facilitating the laryngoscopic examination.

After various attempts to catch the wart, I succeeded, about the middle of March, in pulling off three small granules from its apex, Mr. French, of Great Marlborough Street, being present on the occasion. A few days afterwards I found that the wart had shrunk more than could be accounted for by the small pieces I had pulled off; and the voice was decidedly stronger.

On three successive occasions, at intervals of three or four days, I made an unsuccessful attempt to catch the wart; on each occasion I touched it with a brush dipped in a solution of nitrate of silver, ℈j to ʒj, and on each occasion it was evident that the wart was dwindling away, until on the 11th April I found it had entirely disappeared, leaving only a very slight smooth thickening of the cord where the wart had been attached. He now said that his voice was nearly as strong and as clear as it had ever been. One of the

greatest inconveniences experienced when the wart was present was that, in consequence of the windage caused by the partially open state of the glottis in speaking, the air escaped so rapidly from the chest that he was out of breath before he could finish a sentence. This source of distress has now entirely passed away, and he can read and speak with comfort. More recently he tells me that he has taken the chair at a club dinner, involving much after-dinner speaking; and that he has made a speech at a public dinner entirely to his satisfaction.

I have heard lately that his voice continues strong and clear.

[Mr. White, the subject of this case, attended the meeting of the Society when this paper was read, and, addressing the Society in a clear voice, gave an account of his malady and his recovery.]

Case 6.—W. A—, æt. 8, was an out-patient of King's College Hospital, when I was asked to examine him to ascertain the cause of hoarseness which had existed from his infancy. After several unsuccessful attempts to get a view of his larynx, attempts which were rendered very difficult by the small size and the irritability of his throat, I at length saw plainly a warty growth attached to the anterior extremity of the right vocal cord. The growth was of a somewhat elongated shape, and occupied about one third of the opening of the glottis. His father stated that, when a baby, the child could not cry like other children, and that his breathing was difficult, and attended with noise. He was told that these symptoms would pass away as the child grew. At three years of age he was worse, his breathing being more noisy and difficult, and he was admitted into an hospital where tracheotomy was contemplated but not performed. After a time he left the hospital, apparently somewhat improved. After a few weeks he again became worse, and was taken to another hospital where a blister was applied to the neck, the child crying the whole time that it

was on. The blister was kept open for three weeks by an irritating ointment. Then he had strong emetics, iodine was applied to the neck, and he took "pailsful of cod-liver oil."

Another surgeon who saw him, finding that his tonsils were large, had them cut. Meanwhile, as he grew older, his breathing had become less noisy and difficult, but the voice remained a mere whisper.

In May, 1867, I removed nearly the whole of the growth, and his voice has since been greatly improved. A portion of the growth, however, still remains, and this gives his voice a somewhat reedy character. I hope ere long to complete the cure of this case, but there are several impediments in the way. The remaining portion of the growth is so small that it can with difficulty be caught by the wire, and it will probably have to be destroyed by caustic. Then the boy lives at a distance, and his voice has been so much improved, that he and his father are somewhat indifferent as to any further treatment. His father writing to me lately says, "I am glad to say his voice is much better and stronger, and should it get no stronger, I don't think it would be any denial to him in getting his living."

Case 7.—A gentleman, æt. 48, came to me on the 8th October, 1867, and requested me to examine his larynx before he told me his history. I found that he had a wart about the size of a pea, and of a red colour, on the right vocal cord at the junction of the anterior and middle third of the cord. The wart projected against the opposite cord when he vocalised, so that there was considerable windage, and his voice was husky. He then told me that he had had an uncomfortable feeling in his throat on and off for twenty years, but his voice had been clear and strong until May, 1866, when, as he said, "while he was uttering a loud 'No' in the House of Commons his voice suddenly cracked." A little blood came into his mouth; for several days he was very hoarse, and since that time his voice has been constantly more or less feeble and husky. In May, 1862,

Dr. Bence Jones had taken him to Professor Czermak, who was then staying in London. Czermak discovered what he called a "small polypus," and made a sketch, which the patient showed me, representing the growth in the same situation and of nearly the same size as I now saw it. An interesting question now arose, namely, why a growth upon the vocal cord which had existed for years had rendered him hoarse only since the so-called "cracking" of the voice in the House of Commons eighteen months ago? The most probable explanation appears to be this: up to that time the growth was so placed that when the glottis closed in vocalising the wart was *below* the opposed surfaces, and so the voice was unaffected, but while he was uttering the loud "No" on the occasion in question, the strong expiratory current of air drove the wart upwards between the cords, thus making a sudden gap in the glottis, and instantly changing the sound of his voice. At the same time the growth was stretched and partially torn, as shown by the appearance of blood in the mouth, and henceforth it was so elongated and moveable, that during vocalisation it was always interposed between the cords. The voice, therefore, from that time had been more or less thick and feeble.

This gentleman had been examined, shortly before I saw him, by another laryngoscopist, but no attempt had been made to remove the growth. I told him that sooner or later, I could not say how soon, the tumour might probably be removed by the wire instrument, and he agreed that I should make the attempt. On the 11th October I made several unsuccessful attempts, but as his throat was large, and he was very steady, I felt that sooner or later I should succeed. On the 12th October, I twice caught the growth and pulled off two small pieces. On the 13th, I removed a large piece, which I at first believed to be the entire growth, but I afterwards found that a rounded nodule was left. I made no further attempt until the 4th November, when I caught the wart twice, and each time a piece came away in the wire. Still there was a nodule left about the size of a No. 4 shot. Again the patient went into the country, and I had no

opportunity to repeat the operation until the 1st January, 1868. On that day I made an unsuccessful attempt to catch the wart; but on the following day, after three or four misses, I caught the growth at its junction with the cord, and removed it entire, leaving upon the cord only a slightly uneven surface where the tumour had been attached. Immediately the strength and clearness of his voice were increased. On the 6th January he called to report himself all right. His voice was loud, clear, and ringing. No trace of the growth was to be seen. The portion of the cord from which the growth had been detached appeared somewhat bulging, but its surface was quite smooth.

I had a note from this gentleman, dated March 20th, 1868, in which he says "I am happy to say that my voice is all that I could wish. You may put me down as a perfect cure."

It will be seen then that, in four cases out of the seven, the cure has been complete, and the voice has been restored to its original strength and clearness. In two other cases the restoration of voice has been nearly complete, while in one case, although the morbid growth has been almost entirely removed, there is still considerable feebleness and huskiness of voice in consequence, mainly, of a general roughness of the vocal cords, which prevents their close apposition.

In all the seven cases the morbid growth sprung from the true vocal cord on the *right* side.

In five cases out of the seven, the morbid growths have been preserved and may be inspected by the Society. In case 2, only a portion of the membranous wall of the cyst has been preserved as a microscopic specimen. In case 5, the small portion of the warty growth which was removed by the wire was accidentally destroyed after its removal; the remainder of the growth was destroyed *in sitû* by the application of caustic.

On microscopic examination, all the warty growths appear to consist almost entirely of modified epithelial cells.

The drawings were made for me by Dr. Westmacott, from rough sketches by myself. (See Plate III.)

REFERENCE TO PLATE III.

Fig. 1.—Case I, J. W—.

„ 2.—Case II, Mr. F—.

„ 3.—Case III, Mr. J. G. C—.

Fig. 4.—Case IV, Mr. J. L—.

„ 5.—Case V, Mr. J. W—.

„ 6.—Case VI, W. A—.

Fig. 7.—Case VII, "A gentleman."

Fig.1.

Fig.2.

Fig.3.

Fig.4.

Fig.6.

Fig.5.

Fig.7.

A CONTRIBUTION

TO THE

SURGICAL PATHOLOGY

OF

SERO-SANGUINEOUS CYSTS IN THE NECK AND AXILLA.

BY

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SURGEON TO GUY'S HOSPITAL.

Received April 13th, 1868.—Read June 9th, 1868.

THE cases, the details of which I am about to relate, belong to the class of cystic tumours. That is to say, the tumour consists of a fibrous cyst or capsule which contains serous fluid. The principal cyst may be associated with numerous smaller cysts, with which it is sometimes in direct communication. But in other cases this is not observed.

The serum, coagulable by heat and nitric acid, sometimes contains a constituent which coagulates spontaneously after the fluid is cold. The colour of the pure serum is a pale amber tint, but it varies between dark yellow, brown, or red according to the quantity of blood mixed with it; and, as will be observed hereafter, it varies at different times when taken out of the same cyst.

This brief reference to facts will suffice to identify the cases to be related with those termed "Hydrocele of the

neck" by some authors, "Sero-cystic disease" by others, and Hygromata by foreign pathologists.

The first recorded case which I have been able to find was published by Damen at the Hague, in 1780. The illustration accompanying it is interesting in relation to one I shall describe, and which is doubtless a repetition of it at the early stage.

J. P. Maunoir was the next surgeon who called attention to these cases, under the name of "Hydrocele of the neck;" since then German, French, and English surgeons have, from time to time, added to our knowledge of this disease under various designations.

Mr. Lawrence and Mr. Cæsar Hawkins have contributed to the 'Transactions' of this Society, cases which they have observed; but in consequence, probably, of the rarity of the disease, little knowledge has since been acquired.

Some cases were published by myself in 'Guy's Hospital Reports' for 1860; Mr. Holmes described some in 'The Lancet,' in 1864; and Mr. Thomas Smith, in 1866, recorded some cases of congenital cysts cured by the introduction of a single thread seton.

The only complete monograph on this disease was published at Berlin in 1855, by Dr. E. Gurlt, who, with true German erudition, has quoted a large number of cases ('Uber die Cystengeschwülste des Halses,' Berlin, 1855).

The following examples occurred in my own practice, and, with the exception of the early history of Case 3, have never been published.

Case 1.—In April, 1862, a healthy, temperate man, æt. 28, was admitted into Guy's Hospital under my care. He was a native of Pembrokeshire, and by trade a joiner.

Five or six years before, when about twenty-two years old, he accidentally felt a small lump in the right axilla, which, during its slow growth, had never caused pain.

In that region there was a very moveable tumour which glided away from direct pressure, and could only be distinctly seen when fixed by compressing the surrounding structures

methodically. The growth seemed to slip away into the subscapular region, between the border of the latissimus dorsi and the ribs. When fixed, the integuments were irregular over its surface with elevations and depressions exactly resembling the appearances produced by a fatty growth. Such, indeed, was the diagnosis of the tumour given by all who examined it. The integuments were quite healthy. The man readily assented to its removal.

I made a vertical incision over the centre of the tumour parallel with the anterior border of the latissimus dorsi muscle, and exposed, instead of fat, a mass of cysts with fluid serous contents. With the finger, the handle of the scalpel and now and then its cutting edge to divide the more resisting fibrous connections, I had isolated the larger part of the growth when a cyst burst, about three or four ounces of amber-coloured, clear serum escaped, and the walls collapsed. After this, the growth when removed looked like a mass of fibre tissue. One artery was secured which ran across the growth and accompanied an intercosto-humeral nerve which was divided. The bleeding was insignificant. The wound was dressed an hour after the operation, and it quickly healed, although more serous fluid ran from it than usually happens after ordinary wounds.

The new growth consisted of a cyst with bulging sides, forming cystiform projections (see Plate IV). The elementary fibre tissues were the contractile and filamentary. I attempted to distend the cysts by injecting spirit and water, but in consequence of the large quantity of the contractile fibre element they soon collapsed. Their cavity was lined with a smooth, bluish white serous membrane. The preparation from which the drawing was made was obtained by stuffing the cysts and hardening the tissues in dilute spirit.

Case 2.—A healthy boy, æt 7, was brought to me in October, 1860, on account of a tumour which occupied the right axillary and subscapular regions.

When he was *a year old* a small lump was accidentally felt on the right side of the chest, below the clavicle, midway

between the nipple and shoulder. The integuments were not affected. It slowly increased and, four years after first observing it, a puncture was made, and about three fourths of a pint of colourless fluid removed. The cyst refilled, and in February, 1859, about the same quantity of serum as above described was withdrawn. Neither of these operations was followed by any constitutional disturbance. The fluid again collected, and when I next examined the case the following conditions existed.

There was a large swelling occupying the right axilla, extending forwards to the mammilla, and backwards between the scapula and thorax. It was easily pressed in every direction, and, when the arm was brought against the side of the chest, the swelling bulged forwards and backwards. The wall of the cyst was not very tense, so that its thickness could be determined by nipping it up between the finger and thumb. The wavy, vibratory movement of a circumscribed collection of fluid was visible and palpable throughout the swelling, when its surface was gently tapped with the fingers. The translucency of the swelling was very apparent. The patient never experienced any pain in the part. It was easily ascertained that the cyst extended beneath the pectoral muscle in front, and between the scapula and the thorax behind. Above it reached the clavicle, between which bone and the tumour the axillary vessels and nerves were situated.

1st Operation, October, 1860.—I performed paracentesis of the cyst, and removed about twelve ounces of amber-coloured, clear fluid in which laminæ of cholestearine floated. Heat coagulated it into a solid mass.

A second very small cyst was felt above and attached to the collapsed one.

2nd Operation, September, 1861.—At this date the swelling being inconveniently large, I removed about three ounces and a half of serum possessing the characters above described. When the cyst was collapsed, I could feel a solid mass in the armpit. On this account I delayed injecting the cyst with tincture of iodine, the treatment I had proposed to adopt.

3rd Operation, March, 1862.—In March, 1862, four ounces of serum of dark brown colour were removed. After standing and becoming cold, there appeared in a few hours a spontaneous formation of a fibrinous mass which entangled a few blood corpuscles, and was, therefore, of a faint pinkish tint. A solid mass, larger than I had before felt, remained in the axilla, the precise composition of which I was unable to ascertain. Considering that iodine injections would fail to eradicate the disease, I now proposed to cut it out.

After a consultation with Mr. Cæsar Hawkins, Mr. Paget, and Dr. Harcourt, the medical attendant of the family, at which the perfect innocence of the growth was recognised, it was decided to delay all active treatment for a time.

The cyst soon refilled again, and the size of the tumour being troublesome I proposed to excise it. The consent of the parents of the child having been obtained, I undertook the operation on October 1st, 1862. The patient being fully under the influence of chloroform, I made a single linear incision along the prominent surface of the cyst, from above downwards, the lowermost border of that structure being first cleared of the surrounding tissues. Each arterial vessel was tied with silk as soon as cut. They were generally small but numerous. Having reached the solid part of the tumour, which was deeply seated in the axilla, it was found more easy to dissect it out after having emptied the cyst; although the cyst itself was dissected from its adhesions when full. As soon as the tumour was completely detached from its fibrous connections it appeared as if the external respiratory nerve ran through it, for it hung by it only. However, by careful dissection this important nerve was detached from its posterior surface without injury; although, at first sight, it appeared to be deeply imbedded in its substance. The axillary hollow, its muscles, vessels, and nerves were all clearly exposed when the tumour was removed from it. Only a few minute filaments of the intercosto-humeral nerves were cut, as was subsequently discovered by numbness of small patches of skin on the side of the chest. The operation occupied more than an hour.

Progress after Operation.—He suffered very much from chloroform nausea, and after a slight feverish attack on the third day a vesicular eruption appeared on the right upper extremity, which in a few days became pustular and then dried off. A great quantity of serous fluid ran from the wound, which was dressed with lint, the edges being held together by adhesive plaister. At the end of fourteen days all the ligatures had been taken away and the boy was convalescent. On the sixteenth day from the operation he left London, the wound being nearly healed. Slight suppuration appeared after this, but in a month the wound was perfectly cicatrized.

At the date of writing this history, more than five years having elapsed since the treatment of the case, there is no reappearance of the growth, and the patient is robust and healthy.

Description of the Tumour (see Plate V, fig. 1).—The mass removed consisted of the principal cyst, a more solid portion, numerous lymphatic glands with their vessels, a large quantity of connective tissue and fat.

1. The cyst was very thin and, when filled with fluid, more bulky than the rest. The largest part of its outer surface was loosely connected with the collateral muscles, blood-vessels, and nerves; but as it approached the solid part of the tumour, to which it was closely attached at one end, thin outlying portions of a new growth, consisting of cells, were firmly united with it. The surface of its lining membrane was uniformly smooth, although streaked here and there with lines resembling fine cicatrices. I could not demonstrate any distinct communication between this large cavity and the cellular structure composing the more solid portion.

2. The cellular division was about one third the size of the cyst, when filled, and circumscribed by a delicate fibrous envelope. Sections display its remarkable cellular composition. The whole of it is broken up into innumerable cell-spaces communicating with one another, the membranous walls of the cells being as delicate as gold-beater's skin.

Several lymphatic glands and their vascular plexus were

firmly united to the growth, the largest of which is seen on the preparation; the rest were removed.

CASE 3.—A healthy-looking, well-formed man, æt. 20, was admitted into Guy's Hospital under my care in December, 1867, on account of a large tumour occupying the right side of the neck and axilla. It was painless and simply inconvenient from its bulk. Even the movements of the shoulder joint were not embarrassed.

Soon after birth his mother observed a small tumour in the neck just *above* the right clavicle, about the size of a horse bean. He was a remarkably healthy child. The swelling increased, and by degrees descended behind the clavicle and appeared in the axilla.

I saw this boy in 1850, then three years old, with Mr. Hugh Rump, of Wells, Norfolk. At my suggestion a trocar and canula were inserted at its lower part, and about nine ounces of serum flowed out. The fluid was clear, limpid, of a pale amber colour, and contained a material which coagulated shortly after being withdrawn. The region in which the tumour was situated was strapped over with adhesive plaister, a compress and bandage being placed in the axilla and over the shoulder. Severe constitutional effects were produced by this operation. Great prostration ensued on the second day after this simple operation, and for a short time life was in jeopardy. The child appeared to be in the condition of a person suffering from hæmorrhage, although *not a drop of blood was lost* during the operation. The failing health of the child necessitated the removal of the bandages in about a fortnight from the operation, which being done, the cyst was found to be refilling, and in a short time it was quite as distended with fluid as it had been prior to the operation. The child, however, recovered, and I heard no more of him until the autumn of 1867, when he entered Guy's Hospital in the hope of having the tumour removed.

The accompanying illustrations (see Plates VI and VII) preclude the necessity of describing the external configuration of the tumour at that time. To the touch it was clear that

the contents were fluid. Pressure on the supra-clavicular tumour made that one below the clavicle very tense, and *vice versâ*. The clavicle was traceable in front of the swelling, and the axillary artery could be felt passing along the front of it. The right radial pulse scarcely differed from the left.

Judging from the previous history of the case, that the tumour was composed of a cyst filled with serum, I proposed to reduce its dimensions by repeated paracentesis, in the hope that when confined within smaller limits I might be able to excise it.

The first puncture was made in the tumour at the outer and lower part, where a slight furrow appeared below the border of the pectoralis major. Only fluid containing a large proportion of blood flowed out. It appeared at first sight, indeed, to be pure blood and of a bright red hue like the colour of arterial. This surprised me. However, twenty-three ounces were removed without any constitutional effect arising, although the man was carefully watched. He was not under the influence of chloroform.

At intervals of four days, six, three, and again four, from each separate operation, twenty-three fluid ounces, forty, fifty, and forty-five were drawn off through a canula. So that in a period of seventeen days, five punctures having been made, 181 fluid ounces, or $1\frac{1}{8}$ gallons of the fluid, were abstracted. The punctures were made at different parts of the walls; two below the clavicle, three above it. Not the slightest constitutional disturbance followed either operation. The tumour remained for some time more or less flaccid after each, and after the evacuation of fifty ounces, that division of it above the clavicle almost disappeared. But the cyst slowly refilled always.

The characters of the fluid, alike on every occasion, were as follows:—

First operation—quantity, fl. oz. 23; sp. gr., when warm, 1028; sp. gr., when cold, 1035.

There was no coagulation after twenty-four hours' rest. The red globules had subsided and left clear serum in the

upper part of a graduated glass measure in the following proportions:

Of serum, fl. oz. 10: sp. gr., cold 1016. Of red globules and serum, fl. oz. 13: sp. gr. cold 1050.

The serum was of a dark-brown colour with a greenish hue when examined with the light shining upon it. It solidified entirely by heat and acid.

The blood-corpuscles arranged themselves into rolls when the warm fluid was examined under the microscope, exactly as they do in ordinary blood, and they appeared to be well shaped, generally.

This patient was in the hospital twenty-nine days, during which time he was allowed nourishing diet, and tonics were administered. But he evidently lost strength, although he was not disposed to admit it. His pulse became weak; his cheeks and lips pallid; and he was not so fat as when admitted. He was very anxious, however, that more active measures should be taken to effect a cure, and, although I explained to him that great risk to life would attend any attempt to do so, I believe he would have submitted to any procedure. But at this conjuncture he was required to return to the country on domestic affairs.

I desire now, as briefly as possible, to invite the attention of the Fellows to a few inferences suggested by the facts adduced.

First, as to the true nature of the growth in the first and second case. There are writers who advocate the doctrine that the serous cysts of the neck and other parts "appear to be transformations of vascular tumours" ('Lect. on Surg. Path.,' by James Paget, F.R.S., vol. ii, p. 38). That opinion was first expressed by Mr. Lawrence in a clinical lecture on congenital cystic tumours removed by him from the thigh, neck, trunk, mouth, and lips. Nearly at the end of the lecture we read, "I merely set before you the conclusions deduced by Mr. Coote," who made "a careful examination of the parts removed in the cases now related" ('Med. Times,' 1850, pp. 559, 561). Now, if the conditions stated by Mr. Paget to exist "of the close connection which some of these cysts have

with large deep-seated veins, the occasional opening of blood-vessels into their cavities, and their sometimes distinctly forming portions of vascular nævi" (Paget, vol. ii, p. 38) be taken as conclusive evidence that these congenital cystic growths are to be regarded as "transformations" of the tissues forming nævi, or of erectile structure, these two tumours cannot be placed in the category of such growths. Because there were no large blood-vessels in connection with the cysts, nor was there any portion of a vascular nævus attached to either of them, the cystiform structure which a superficial observer might regard as identical with that of a nævus is really not so.

True nævus structure so intimately resembles that composing the corpora cavernosa penis as scarcely to be distinguishable from it; and, moreover, enjoys great vascularity like it. Now, the structure of the cellular part of both the tumours, cases 1 and 2 (see Plate IV and V, fig. 1), does not resemble either the one or the other.

To afford the members of this meeting an opportunity to form their own judgment on this anatomical point, sections of a subcutaneous nævus (Plate V, fig. 2), of the corpora cavernosa, and the tumour, No. 2 (Plate V, fig. 1), are suspended in a glass jar for comparison.

The nævus tissue shows a very fine, delicate, reticular, arrangement of fibrous trabeculæ, just like that of the corpora cavernosa; whilst the section of tumour No. 2 displays thin lamellæ of fibre tissue, perfectly different in form and disposition to either of the other sections. No two structures could well be more dissimilar. Further, every surgeon knows the peculiarity of the bleeding which follows an incision of a nævus; no such bleeding ensued in either of the cases described. In the first growth there was no portion resembling nævus in the slightest degree. In a true subcutaneous nævus, removed by myself from a child's back, immediately one of the collateral veins was cut the whole growth collapsed, and I was enabled to make the preparation exhibited by injecting spirit into it from that vessel.

The conclusion, therefore, which I am disposed to adopt is

that this cystiform growth should be regarded as a peculiar development of its own kind and individuality; basing this opinion upon its structure and vascularity, both of which are so remarkably dissimilar from any nævus I have examined.

At the same time, I would not deny that genuine nævi may undergo those changes described as cystiform; that they may retain intimate communications with neighbouring blood-vessels, or, that large veins lying contiguous to them may be cut during the operation for their removal.

Secondly, as regards the treatment of the cases by excision of the tumour. The repetition of the operation of paracentesis of the cyst having failed to effect a permanent cure of the tumour in the second case, and the fact being evident that a large growth existed in contiguity with the cyst, rendered it incumbent to adopt some method that would ensure the effectual eradication of the complaint.

I objected to the use of irritating injections on account of the existence of the more solid growth, which I thought would not be cured by that means, and the removal of which, by a cutting operation, if it should be required, might be made more difficult after the inflammation excited by them. Besides, the probable event of prolonged suppuration, causing great depression of the vital powers, was not overlooked in the consideration of the case.

Judging from the results of the operation performed in cases 1 and 2, the age of the two patients being considered, the method adopted seems to have been the correct one. Other plans of treatment would, doubtless, be preferable in infancy and early childhood, of which the cases published by Mr. Thomas Smith, before referred to, are good illustrations.

The accompaniment of numerous and enlarged lymphatic glands—a fact alluded to by Mr. Cæsar Hawkins and other writers—formed a striking feature in the second case; and their removal, without causing any untoward symptoms, is worthy of a passing notice.

The third case doubtless belongs to the class of congenital serous cysts, as the mother observed the cervical tumour *soon after the birth of the infant.* Its precise nature was

ascertained when the boy had passed his third year, by taking clear pale yellow coagulable serum from the cyst *without* the slightest admixture of *blood*. The next puncture, made seventeen years afterwards, revealed an important addition to the contents of the cyst. The fluid was to all appearances pure blood. But it differed from blood in the absence of its characteristic phenomenon, spontaneous coagulation. Some very interesting observations "On Tumours containing Fluid Blood" have been published by Mr. W. Morrant Baker, in which the causes interfering with its coagulation are ably stated. The limits of this paper will not allow the consideration of this subject here, but the fluid removed from this man agreed probably with that described by Mr. Baker as follows: "The fluid is not simply uncoagulated blood, but a mixture of serum (derived probably, not only from extravasated blood, but secreted also by surrounding parts) with blood-cells, diffused colouring matter, and disintegrated fibrine" ('St. Bartholomew's Hospital Reports,' vol. i, p. 218).

There was no reason to conclude that this cyst was lined with adherent fibrine; on the contrary, it felt, when partially empty, exceedingly thin and membranous.

I have been able to find only two cases resembling this one. That by Damen, already quoted, exactly corresponding in site and outline, was not punctured, therefore we are ignorant of the nature of its contents. Another, recorded by Gurlt, identical with mine as regards locality and form, when punctured, the age of the man being then eighteen, contained bloody serum like it. Therefore, the case I have now related to this meeting I believe to be unique as regards the circumstance of the cyst, containing, in childhood, pure serum, subsequently, in manhood, serum mingled with blood.

The source of the blood is, at present, involved in mystery; whether it flows from a single arterial or venous vessel, or oozes from a parietal growth, are facts only to be ascertained by dissection. The moment when the blood first mixed with the serum is also a subject for conjecture, although the description of the effects produced by the first operation by

Mr. Rump, who narrowly watched the child, and wrote them years before he knew the cyst contained any blood, would justify the inference that the bleeding into the cyst occurred soon after the first puncture was made.

In conclusion, I have merely to add, that I trust the cases now related may contribute to the knowledge of sero-sanguineous cysts as regards their pathology and treatment.

Appendix.

October 12th, 1868.—At this date I am informed by Mr. Rump that the patient has quite recovered his health and strength, his general health being vastly improved since he left the hospital. The tumour which was above the clavicle has now entirely disappeared (by gravitation), but the axillary and subscapular tumours are larger; they produce, however, little inconvenience.

DESCRIPTION OF PLATES IV, V, VI, AND VII.

PLATE IV.

Cystiform growth from the axilla, removed April 15th, 1862, from the right axillary region of a man, J. P—, æt. 28. It had been observed five or six years, and slowly increased without pain. (See page 187.)

PLATE V.

Fig. 1.—Section of the more solid part of the tumour excised from the axilla in Case 2. (See page 190.)

Fig. 2.—Section of a subcutaneous nævus removed from the thoracic region of a child. The trabecular structure is well displayed, and closely resembles that of corpora cavernosa et spongiosum penis. (See page 194.)

PLATE VI.

Sero-sanguineous cyst in the neck and axilla. Case of J. C—, æt. 20. Front view. The cyst, as represented in the plate, was tapped, and at five punctures, in seventeen days, above one gallon of serum mixed with a large quantity of blood was removed, but without any striking results, and the tumour remained of its original bulk. (See page 191.)

PLATE VII.

Ditto, ditto, back view.

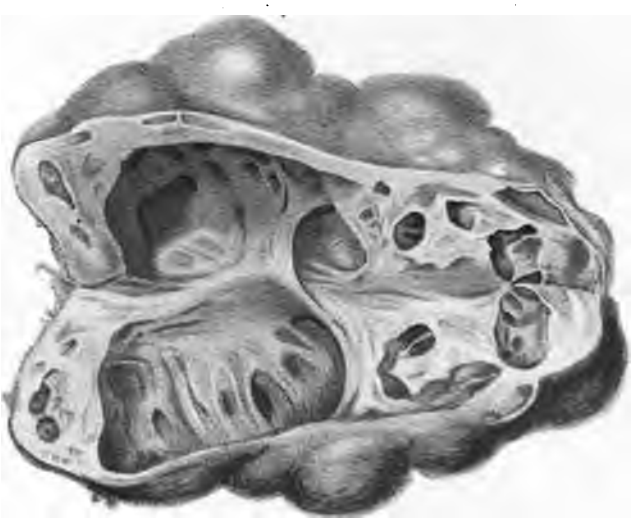

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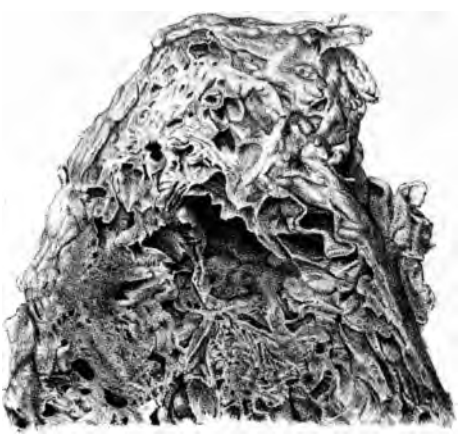
Fig 2.

Fig 1.



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ON THE

TORSION OF ARTERIES AS A MEANS OF ARRESTING HÆMORRHAGE,

WITH EXPERIMENTS.

BY

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Received April 15th.—Read June 9th, 1868.

On the 16th of July, 1829, M. Amussat read before a meeting of the Académie Royale de Médecine a paper on his new method of arresting hæmorrhage from large blood-vessels without the aid of the ligature, the plan having been, as he tells us, suggested to his mind by the long recognised fact that "torn wounds do not bleed."

He supported his suggestions by relating the details of some experiments he had made upon animals, as well as upon the human subject, and quoted four cases of amputation of the thigh, and one of the arm, in which hæmorrhage had been readily and permanently arrested by simple torsion.

His practice was at once adopted in Paris by MM. Boyer, Dupuytren, Velpeau and Thierry: also by Lieber of Prussia, Fricke of Hamburg, and Schrader of Dresden. It received also the unqualified support of Majendie, who described it as one of the most brilliant discoveries in modern surgery.

In the periodicals of the day many cases are also related in which these surgeons had practised torsion of the arteries with immediate and permanent success, even to the divided extremities of large vessels; and amongst these are cases of disarticulation of the shoulder-joint, amputation of the thigh, leg, arm, and forearm; castration, extirpation of the breast, and other tumours.

The surgeons of this country, however, took little or no notice of the practice for some years; and it was not till the year 1834, five years after Amussat's paper had been published, that the subject was clearly brought before the notice of the profession: when Mr. W. B. Costello read before the Westminster Medical Society a paper "On the Torsion of Arteries for the purpose of arresting Hæmorrhage," which was subsequently published in the 'Lancet' of March 8th, 1834.

In this paper Mr. Costello gave the results of M. Amussat's practice, and detailed the particulars of five successful experiments which he had made upon dogs under that surgeon's direction.

The paper was well received, and some notice was taken of it at the time, but the practice was never followed, and, from that day to this, torsion, as a means of arresting hæmorrhage in large vessels, has never been practised, although for small ones it has been occasionally employed. An explanation of this apparent want of attention to this new method of arresting hæmorrhage may possibly be found in the fact that it was suggested before its time; that is, before the introduction of chloroform, by the use of which the surgeon is enabled to take up all vessels with care and precision, and there is no object in hurrying over any of his proceedings—a condition of things which can hardly be said to have existed under previous circumstances, when the surgeon's anxiety was to complete his operation as speedily as possible, and it was a source of satisfaction to know that a stout ligature was on the end of a large vessel.

Within the last few years, however, the attention of the profession has been much directed to the subject of hæmo-

statics; and it would seem that many surgeons have become clearly dissatisfied with the practice generally adopted of ligaturing divided vessels, and of leaving the ends of the ligatures within a wound. It being stated, as a broad objection to their use, that they, by their presence, prevent a rapid or primary union of a wound; that they keep up if they do not excite suppurative action within the parts, and thus add to the danger of an operation by postponing recovery and rendering a patient more prone to that fatal scourge pyæmia than he would be under other circumstances; to these another objection may still be added, that the ligatures on their separation from the artery are apt to be followed by secondary hæmorrhage.

To Sir James Simpson unquestionably our thanks are due for having specially directed our attention to this subject, and for having suggested a plan of treatment which, to his own mind and that of others, aims at rendering more simple our mode of arresting hæmorrhage, and at the same time does away with some of the objections which have been adduced against the use of the ligature.

It is not my intention to inquire into this question on the present occasion. It has able advocates in the person of its distinguished author, and in Messrs. Keith and Pirrie; it is, moreover, now under trial by the profession. It has doubtless advantages over the ligature, and disadvantages which have to be comparatively examined by the grand test of experience, by which it will stand or fall. I have practised it myself, and seen it practised in many ways, but regret to say that neither my observation nor experience have been of the most satisfactory kind.

It has, however, physiologically considered, one point of weakness which has some bearing on my present subject, for the value of the practice of acupressure rests on what has hitherto been looked upon, and what must still be described, as being a temporary mechanical obstruction to arterial hæmorrhage; the permanent arrest of bleeding depending in this practice, as in others, on natural processes; now, in acupressure the permanent arrest of hæmorrhage depends

entirely upon the clot which forms in the vessel, for no evidence has yet been adduced to show that any change occurs in the inner tunics of the vessels which have been subjected to the pressure of an acupressure needle, such as is well known to take place after the use of the ligature, and such as also occurs after the application of torsion in large vessels. In these last two forms of practice the two inner coats are divided, and subsequently unite, the permanent arrest of hæmorrhage depending materially on such a process; in acupressure there is no evidence whatever to show that any such union takes place between the two surfaces of the vessel which are brought in contact; as a consequence the permanent success of acupressure depends upon the coagulating power of the blood in the occluded vessel, the needle acting as a temporary mechanical obstruction to the flow of blood during the brief period of its presence.

Physiologically, therefore, acupressure is an uncertain process, and when contrasted with the ligature and torsion stands condemned; for its success depends only upon the temporary processes which nature adopts for the purpose of arresting hæmorrhage, and not upon the permanent hæmostatic changes which nature employs in other cases.

When Professor Syme, therefore, in a short note published in the 'Lancet' of January 4th, drew the attention of the profession back again to the subject of torsion, I must confess to feeling a sense of satisfaction, for it seemed tolerably clear that, if it could be practically proved that hæmorrhage from a divided artery could be safely and permanently controlled by such a method, we should have a means at our disposal which would be entirely free from all the objections which the strongest objectors to the use of the ligature could possibly adduce, and which in its simplicity and value would without doubt bear favorable comparison with acupressure as well as with the ligature.

It was under these circumstances that I was thus led to examine into the question, to try it in practice, and to prove it by experiment; and I now propose to lay the results of my inquiries before the Fellows of this Society, with the hope

that they will be induced to test the practice for themselves, and by the aid of experiment gauge its value.

I propose to relate seriatim the experiments I have made upon the dog, horse, and human subject to test the value of torsion, and to observe the process by which the vessels so treated become permanently sealed; having previously described the two methods by which torsion has been practised.

I shall pass on to describe the physiological changes which have taken place in the arteries which have been twisted; and draw attention to such special points as it seems necessary to attend to in the application of the practice. Some brief comparison will then be made between the value of torsion and the ligature, and some general deductions drawn up from the consideration of the subject as a whole.

I must add that in most of my experiments I have had the benefit of Mr. H. Howse's able assistance; that he has given me valuable aid in examining the vessels of the animals after death, and that the descriptions of the physiological conditions of the arteries are chiefly from his pen, receiving from me only such slight alterations as I deemed necessary. I am also indebted to him for the drawing of some of the preparations.

It would be well, however, *in limine*, to premise that there are two modes of applying torsion; one described by Amussat as "free" torsion, the other as "limited."

In *free torsion* the end of the artery should be fixed by a pair of clasp forceps and twisted freely.

In *limited torsion* the artery, having been drawn out of its sheath, should be fixed *transversely* about three quarters of an inch from its divided extremities by a pair of clasp forceps and held steadily; whilst with a second pair the free end of the vessel should be twisted freely as in the former, the object of the first pair of forceps being to fix a limit to the twisting of the vessel and prevent the artery being separated for any distance from its vascular attachments.

Experiments on the Dog.

Exp. 1.—February 4th, 1868.—I divided the left *femoral* artery of a dog just below Poupart's ligament, and twisted the cardiac end by "free" torsion *four* times with success. During this time the distal end was held by forceps, and when these were removed hæmorrhage occurred; the bleeding extremity was, however, seized by forceps and twisted *four* complete revolutions, all bleeding at once ceased, and by the seventh day the wound had united.

The dog was killed on the eleventh day after the operation.

Exp. 2.—February 8th, 1868.—I divided the right *femoral* artery of a dog and twisted its cardiac end *three* times by "free" torsion; hæmorrhage, however, at once occurred, the end was accordingly twisted *four* times more, when all bleeding ceased. *Four* twists to the distal end of the vessel at once succeeded. On the third day the dog appeared as if nothing had been done to him. The wound looked very healthy.

It must be noted that the blood from the lower part of the vessel came out quite black, like venous blood; the contrast between it and that from the cardiac extremity was very marked.[1]

The dog was killed on the seventh day.

Exp. 3.—February 8th, 1868.—I divided the left *femoral* artery of a dog high up, and twisted its cardiac end *three* rotations with success, not a drop of blood escaping. The same treatment was also applied to the distal end, with a like result.

On the third day the dog was well, and the wound healing.

The dog was killed on the tenth day.

[1] This fact has been observed in the human subject, particularly by Guthrie, but is not generally recognised. It does not appear, however, to be constant.

Exp. 4.—February 11th, 1868.—I cut down upon and divided the right *common carotid* artery of a dog. I applied "free" torsion to its cardiac end, making *three* revolutions, without success, and accordingly seized the vessel again and twisted it four times more. Hæmorrhage was at once arrested. *Three* complete twists were then given to the distal end of the artery, and no bleeding followed.

On the second day the dog was quite well, he had taken his food as usual, and appeared in no way disturbed by the operation. On the following day the animal was destroyed.

It must be noticed that in this case, as in the second experiment, three rotations of the artery were not sufficient to arrest bleeding; four proved successful in both cases.

Both the distal and cardiac ends of this artery have been drawn (see Plate VIII, fig. 2).

Exp. 5.—February 11th, 1868.—Cut down upon and divided the right *common carotid* artery of a dog. Twisted the cardiac end *four* times, with immediate success, and likewise the distal end. Hardly a drop of blood was lost. On the second day the dog appeared to be as well as he was before the operation. On the fourth day the wound had nearly healed.

The animal was destroyed on the seventh day.

Exp. 6.—February 15th, 1868.—I cut down upon and divided the left *common carotid* artery of a dog. I applied free torsion to the cardiac end of the vessel, making *four* complete revolutions of the forceps; all hæmorrhage ceasing. The distal end was firmly pinched by a pair of forceps both at its free extremities as well as one inch up the artery; no bleeding took place.

The animal was killed on the third day.

Experiments on the Horse.

Exp. 7.—March 17th, 1868.—I cut down upon and divided the left *common carotid* artery of a horse; applied two pairs of torsion forceps transversely to the vessel, and divided the artery midway between them, leaving an inch of artery on the distal side of each pair of forceps. With a third pair of torsion forceps I then seized the extremity of the artery at its cardiac end, and twisted it *seven* complete revolutions. I then removed the instrument that fixed the vessel, and not a drop of blood escaped; the pulsations in the vessel were very strong. The same treatment was then applied to the distal end with a like result. It was certainly somewhat astonishing to see the great vessel fill out and pulsate after the operation without one drop of blood escaping; and although the animal plunged somewhat during and after the operation, the success was most complete.

The animal was allowed to live for forty-eight hours, and then killed.

The condition of the vessel will be described in another page (see Plate VIII, fig. 3).

Exp. 8.—April 2, 1868.—I cut down upon and divided the left *common carotid* artery of a horse. I applied a *ligature* to the distal end, and *twisted* the cardiac, employing in this instance, for a definite object, a different method to that which I had used in the former experiment. In both, about one inch of the divided artery was left beyond the point at which the vessel was fixed by the forceps, which were applied transversely; but in the former successful experiment the orifice of the vessel was alone seized before the torsion was applied; in the latter, the extremity of the vessel was taken hold of nearly its whole length, the twisting of the artery being confined to the spot at which it was held; in the one case about one eighth of an inch was allowed for the purposes of torsion, in the other half an inch. *Three* complete twists were alone given, but by those the external coat was so

twisted as to appear hardly larger than a crowquill, and I must confess to feeling somewhat alarmed lest I had ruptured the vessel. On removing the forceps that fixed the artery no bleeding took place, and I hoped all would have been well; within a minute, however, signs of bleeding occurred, and on opening the wound it was clear that blood escaped from a small laceration of the external coat at the point of twist, the twist having remained unaltered. It being apparent that the violence of the torsion had been too much concentrated at one spot, I accordingly seized the vessel lower down, cut off the injured end, and applied limited torsion as in the last experiment, holding the orifice of the artery alone, and giving *six* complete revolutions of the forceps to the free inch of the vessel. No bleeding followed the removal of the instrument, complete success having been secured.

The horse was killed on the fourth day—eighty-seven hours after the operation. No single bad symptom had shown itself. The animal had taken his food ravenously during the interval.[1]

The appearances found on dissection will be described further on.

Mr. Towne has made a model of this preparation, showing the physiological changes which have taken place in the artery after the application of a ligature and torsion respectively.

The design for the model was shown when the paper was read, and a drawing from it (see Plate IX).

Effects of Torsion on the artery of a patient to whom it was applied during life.

I have only one observation to make bearing upon this point, but it is of value, for it well supports practically and physiologically the results of the experiments I have already given upon the horse and dog.

[1] In these experiments on the horse I had the kind and able assistance of Mr. Moss, M.R.C.V.S., of Vauxhall.

On January 13th, 1868, I applied "free" torsion to the brachial artery of a woman, æt. 68, for whom amputation was demanded in the upper third of the arm for a compound comminuted fracture of the elbow-joint, and extensive laceration of the soft parts. The artery was twisted *four* revolutions by "free" torsion, one blade of the forceps being introduced at least half an inch into the vessel, and no bleeding followed, although the pulsation of the artery was very strong. The patient died thirty-five hours after the operation from thoracic complications, and thus I had an opportunity of examining the changes which had taken place in the vessel. I should add that some bleeding took place from the stump twelve hours after the amputation had been performed, from a vessel which had not been seen at the time; and, to this, torsion was successfully applied by the House Surgeon, Mr. Morris, who stated that when the stump was opened, the brachial artery was seen to be pulsating powerfully, but not a drop of blood escaped.

The changes found in the vessel will be described with the other cases, and seen in a drawing (see Plate VIII, fig. 1).

A DESCRIPTION OF THE PHYSIOLOGICAL CONDITION OF THE ARTERIES WHICH HAD BEEN SUBJECTED TO TORSION, AS SEEN IN THE BRACHIAL ARTERY OF THE HUMAN SUBJECT, AND SEVEN FIRST EXPERIMENTS IN ANIMALS.

Mode of preparation.—The vessels were all hardened in spirit immediately after removal from the subject, and then longitudinal sections cut.

Condition of the human brachial artery.—The artery was removed from the innominate downwards. The aorta and innominate were very atheromatous, but this did not extend to any very marked degree to the brachial.

The diameter of the artery was about a quarter of an inch where the clot existed; of the cavity about one sixth of an inch; elsewhere not quite so much. The twist, which was quite local, though free torsion had been practised, was about an inch from the amputated extremity of the artery. The

distal end of the artery was empty. The *cardiac* end contained about two inches of clot; where this ceased a small artery came off. The middle and internal tunics of the artery had clearly ruptured and retracted, they also were completely *incurved* as seen in Plate VIII, fig. 1, meeting in the middle line of the artery; beyond this spot was a small quantity of firm defibrinated clot, which would serve to maintain the incurved tunics in position, and which was covered by the twisted external coat. At the *distal* end of the artery the middle coat was not incurved, the ends merely coming into contact in the middle line.

Experiments on the Dog.

To save repetition, it may be stated that in all this series of experiments, although free torsion was performed, the twist in the vessel was found to be localised to a spot a little beyond the bite of the forceps, never spreading up the artery as described by Amussat.

Exp. 1.—Left femoral artery examined on the eleventh day after the experiment.

The *cardiac* end was pervious to rather more than half an inch from the point operated on. The parietes of the pervious portion were thickened, as if the original, comparatively speaking, thin-walled artery had contracted down upon the cavity. The obstructed part had an ampulla-like appearance, was filled with firm fibrous clot, in which were one or two cavities, containing grumous matter, as if from the commencing softening of the centre of the clot. At the point of torsion, the divided middle coat was perfectly continuous with the other side, so that the cardiac end of the artery formed a perfect cul-de-sac containing fibrin. From the cul-de-sac two branch arteries were given off: one joined the main trunk again beyond the point of operation, the other ended in the surrounding muscles, both were perfectly obstructed by fibrin. Of that part of the cardiac end of the

artery actually seized by the forceps, no trace remained, it seemed to have all atrophied and become absorbed.

The *distal* end of the artery presented none of the appearances above described; it was thin-walled and pervious, nearly up to the point operated upon, where it was obstructed by a small clot which had become much shrivelled.

Exp. 2.—Right femoral artery examined on the seventh day.

The *distal* end was in every respect similar to that of the vessel just described. It was thin-walled, as if the parietes had contracted a little upon the cavity, and was obstructed by scarcely a quarter of an inch of clot.

The *cardiac* end was also like that just described; such complete contraction of the muscular parietes had not, however, taken place in the pervious part of the artery, the wall being thinner and the cavity larger.

The changes in the clot had not advanced so far in either end as in the last experiment; they were not decolorized, nor were there any spots in which it was breaking down.

The part of the artery actually seized by the forceps, although withering, was more distinct in the present case than in the last, but there was no indication of any sloughing of the part in either case.

Exp. 3.—Left femoral artery examined on the tenth day.

Nothing very distinct could be made out in the dissection of this artery, the occlusion and wasting of the vessel had proceeded too far; what remained of it seemed to agree with the other femorals.

Exp. 4.—Carotid artery examined on the fourth day.

The *cardiac* end of the artery was taken out from the innominate. It was obstructed with clot for an inch and a half. The end of the vessel, where the twist had taken place, was dilated into a kind of ampulla, but not nearly so markedly as in the distal end next to be described. The walls of the artery were rather thick, and remained so

to about one sixth of an inch from the twist, where they abruptly became thin from the sudden lessening in diameter of the middle coat, which ran forwards and was lost between the clot and tunica cellulosa or external coat, just at the point of torsion where the ends seemed to rest against a small decolorized plug of fibrin. The part of the artery actually seized by the forceps remained very distinct, though much contracted. The fibrin in the vessel was firm, and with the exception of one or two spots in the ampulla, was not decolorized. A small branch was given off from the ampulla, which was obstructed.

The *distal* end of the vessel was obstructed with clot for the space of three inches. The ampulla-like dilatation in this case was remarkably distinct for half an inch below the point of obstruction; its walls were thin, this being clearly due to the difference in thickness of the middle coat; and its cavity was double in diameter to the cavity of the remaining portion of the artery.

No distinct incurvation of the middle coat of the vessel could be traced.

Half an inch of old withered artery remained above the point where the obstruction commenced.

Both the cardiac and distal ends of this vessel have been drawn (see Plate VIII, fig. 2).

Exp. 5.—Right carotid artery examined on the seventh day.

The vessel seemed in every respect similar to that already described, although the clot was smaller, and the artery seemed more contracted and withered.

The *cardiac* end contained about two inches of clot; where it ceased a small artery came off. The middle and internal coats were completely *incurved,* meeting in the middle line of the artery; beyond this was a small quantity of firm defibrinated clot, which would serve to maintain the incurved tunics in position, and which was covered by the twisted external coat; and, finally, came the distal end of the artery, the middle coat of which was not incurved, the ends merely coming into contact in the middle line.

Exp. 6.—Left carotid artery examined on the third day.

Of the *distal* end of the vessel nothing need be said; it seemed in most respects similar to the others already described.

The *cardiac* end was as usual dilated into a kind of ampulla. The middle tunics seemed to have met and united, but it is somewhat difficult to distinguish them from the fibrin or lymph effused beyond the point of union. The external coat was, however, capable of being dissected from the middle, and there seemed to be some degree of incurvation. About three quarters of an inch of clot had formed in the ampulla and artery leading to it; this was partly decolorized.

Experiments on the Horse.

Exp. 7.—Left common carotid artery examined forty-eight hours after the operation.

Nearly six inches of the cardiac end of the artery were removed, and about two inches and a half of the distal end.

The diameter of the widest part of the *cardiac* end, close by the twist, when in a measure shrunken by spirit, was about three eighths of an inch, the cavity being proportionally large. From the point of torsion, the size gradually diminished towards the cardiac extremity. No branch was given off in the whole of the part taken out.

There was about four inches of continuous clot in the artery from the point of torsion, then came an empty space, and beyond this about three quarters of an inch of semi-detached clot. A piece of fibrin about two inches long, which existed in the middle of the continuous clot, had contracted no adhesion to the walls of the artery; thus it fell out on making the section (see part marked in Plate VIII, fig. 3).

The middle and internal tunics at the point of torsion are turned in and somewhat clubbed, the clubbed ends meeting. Beyond this point comes the twisted external coat; next, a *curious swelling* covered only by the external coat which is

twisted beyond it; and finally, the empty end of the artery where seized by the forceps. The middle coat at this point does not incurve, but merely meets, as in the distal portion of the human brachial artery before described.

The *swelling* already alluded to, which was covered only by the external coat, to the naked eye appeared to contain either defibrinated clot, or lymph; but examined microscopically, it was found to consist of distinct lamellæ of elastic tissue mingled with defibrinated clot. It appears likely, therefore, that it is the middle and internal coats of that part of the artery which had been actually seized with the forceps transversely—for it must be remembered that in this case "limited" torsion was practised—and which had rolled up in this form by their own natural elasticity, or by the twisting; some serum remaining and coagulating in the interstices.

The *distal* end of the artery contained about one inch and a half of clot. The middle coat was turned in and overlapped in the median line, otherwise there was nothing very remarkable in this point.

Exp. 8.—Left common carotid artery examined eighty-seven hours after the operation.

All the soft parts, from the angle of the jaw to the thorax, were removed including the trachea. Directly after their removal, a careful dissection was made, the following conditions being observed.

The *cardiac end* of the artery appeared to be quite empty, it contained only a little liquid blood. At its extremity, where it had been twisted, a mass of lymph existed which had to be dissected off. The twist in the vessel then became very clear. Its free end showed no evidence of having lost its vitality; below the twist a firm clot existed for about a quarter of an inch, and below this a large branch was given off from the vessel.

The *distal end* of the vessel to which a ligature had been applied showed nothing unusual. There was the usual linear division of the middle and inner coats. Both can be well seen in Plate IX, figs. 1 and 2.

On making a section of these parts subsequently it was observed that, in the *cardiac end* which had been twisted, the same changes had taken place as have been already described in other cases. The middle coat had been ruptured and had retracted, its free end having approximated above the small clot which had formed in the vessel. This clot was hardly larger than a pea; at its circumference it was partially decolorized. Below this clot a large branch was given off from the artery. Beyond the clot was the twisted vessel. It was a question whether the small clot in the artery was not placed between the divided inner and middle tunics; this splitting of the tunics taking place after torsion in some cases.

In this case it is tolerably clear that the twisted cellular or external coat must have been the chief obstacle to the arterial flow, the retraction and approximation of the divided inner tunics giving valuable aid. The clot was clearly too small to have much influence, unsupported by the other conditions of the vessel.

On making a section of the *distal end of the vessel* to which a ligature had been applied, it was clear that at the point of ligature ulceration had taken place, nearly separating the end; that the middle tunic of the vessel had been divided and had apparently united. No retraction of this tunic, however, had taken place, nor was any clot found in the vessel.

At the *distal* end of the ligature there was a small clot enclosed by the middle coat, and it will be observed that beyond this point the artery had again contracted, clearly by its own natural elasticity.

To explain the presence of this clot at the cardiac side of the ligature, it should be stated that the ligature was applied to the artery after it had been exposed, and that with my thumb and finger the blood was squeezed backwards before the vessel was fixed by the forceps applied transversely. The artery was then divided and twisted. This clot must have formed at this time, some portion of blood remaining near the ligature and being caught by the divided middle coat.

Experiments upon the arteries of the dead subject.

I have made a large number of experiments on the arteries of the dead subject, healthy and diseased, having generally selected the common iliac and external iliac vessels, and have found that in all the same changes have taken place as have been already described. In every instance the retraction and incurvation of the middle and inner tunics were well marked, and in some the latter was most remarkable; it appears also that in the atheromatous vessels this incurvation is equally well seen.

Indeed this incurvation of the inner tunics of an artery, to which torsion has been applied, appears to be a point of peculiar interest, and from a large number of experiments on the dead subject it may be stated to be a very general result of well-applied torsion.

The amount of incurvation in different instances will, however, be found to vary; in some it will show itself more as an irregular crowding together of the divided and retracted tunics; whilst in others it appears as a complete valvular incurvation of the divided coats, the incurved portions appearing within the vessel as nipple-like projections; under these circumstances, the incurved portions form the most perfect valves it is possible to conceive, being not unlike the semilunar valves of the heart and closing as perfectly. In some cases, again, the middle and inner coats appear to split, and thus to form an additional means for causing coagulation of the blood and obstruction to the artery.

This incurvation is clearly a physical act, and is due to the natural elasticity of the coats of the vessels; it may consequently be confidently expected to take place in all the larger vessels.

The preparations and drawings well demonstrate these points (see Plate X, figs. 1, 2, 3).

I have found also that when a vessel has been efficiently twisted, no legitimate force that can be employed with a syringe, introduced into the vessel above, will unfold the twist in the cellular coat, or undo the retraction and incurvation of

the inner tunics, although the artery will frequently rupture above the part which has been twisted; when the extremity of the vessel has been twisted off, leakage, however, frequently follows.

It should be stated that this incurvation of the inner tunics seems unknown after the ligature; indeed it should be stated that in a large number of experiments which I have made, in which a ligature has been applied to an artery, the only change that takes place in its coats is an imperfect and irregular division of its middle and inner coats; in rare cases this division is very complete, in many others it does not take place at all.

There is never any retraction worth mentioning, and no incurvation. I may refer to Dr. Jones's work on 'Hæmorrhage,' published in 1810, to further illustrate this point.

Résumé of the physiological effects of Torsion on the vessels.

The results of the experiments made upon animals, and the single observation I have had an opportunity of making on the human subject, singularly accord; for in the cardiac and distal ends of the arteries which had been treated by torsion, I found the middle and internal coats of the vessels had been divided; that these coats had retracted in the direction opposed to the blood stream, approximated and overlapped; that at the cardiac end in several instances the coats had become partially incurved, as shown in the preparations and drawings of the carotid of the horse, and the brachial of the human subject; and I found also that every vessel contained clot, from the giving off of the first branch to the point of contact of the two inner divided coats; that lymph or organizable blastema was poured out around the divided extremities of the coats, and between them and the external cellular coat; also that a second smaller clot often existed between the distal end of the two divided inner tunics, and that portion of the cellular coat which had been twisted.

I found, moreover, that the twist which the cellular coat had

sustained was clearly permanent, and that it did not untwist (this can be shown on the dead body). That in no single instance was there any evidence to indicate that the portion of vessel which had been twisted had lost, or was likely to lose, its vitality; or that the parts about the vessel had received any material injury.

That in most cases a kind of ampulla tends to form in the cardiac end of the artery, and sometimes in the distal end. The appearance which this presents varies in every case, and seems to depend on the rapidity with which coagulation takes place in the end of the artery, and upon the amount to which the artery, beyond the point where the clot forms, contracts. In those arteries, therefore, in which much muscular fibre exists, and in which it is healthy, it will be most apparent; thus it is scarcely visible in the brachial artery from probable atheroma, and in the carotid of the first horse which was an old worn-out creature.

It is not necessary, therefore, to suppose that the force of the heart actually dilates the end of the artery. The diameter of the ampulla is in fact something less than the original cavity of the artery. Fibrin forms here first and prevents that close contraction of the parietes which goes on in other parts, and thus an apparent dilatation is formed. The middle coat in the artery is always much thinner than that in the contracted part of the artery (see Plate VIII, fig. 2 B).

Thus the appearance of an ampulla is rather due to unequal contraction than to dilatation.

That though "free torsion" was practised in every case except in the horse, yet the twist in the vessel was local, not extending high up the artery.

That torsion when applied to large vessels immediately above the giving off of a large branch may be successful (see experiment No. 8, on the horse, and Plate IX); and that when applied to diseased vessels in old subjects it may likewise succeed (see torsion of brachial artery, Plate VIII, fig. 1; and preparations of vessels twisted after death, Plate X, figs. 1, 2, 3).

Remarks on the Experiments made on Living Animals.

It is to be noticed that in the experiments upon dogs "free torsion" was employed; in the two upon horses "limited torsion" was used. In all the operation was ultimately successful.

In several on the dog bleeding took place when the torsion had been imperfectly carried out; three revolutions of the vessel being clearly insufficient; in all, however, on the dog four revolutions at once succeeded.

But the arteries of dogs are not large, and although the experiments upon them, as far as they go, must be regarded as satisfactory, they would not be to my mind of sufficient value to prove the power of torsion unsupported by others of greater importance.

The experiments on the horse I cannot but refer to with unmixed satisfaction, for no one can look upon the dilated pulsating carotid artery of such an animal during life, without a feeling of astonishment that such a simple act as that of torsion should be found sufficient to arrest hæmorrhage from its divided extremity; and yet in both the cases related complete success followed the operation when it had been properly performed.

The temporary failure in the second case which followed upon the imperfect application of the practice must be looked upon with favour, for it went to prove what I confidently anticipated would have been the case—that torsion of the external coat when too much concentrated to one spot must end in its rupture, and consequently in failure: but the ultimate success of the torsion when carried out in an efficient manner was most satisfactory, for it tended to show that the means employed were such as could be relied on with some confidence.

The fact that torsion was successfully performed on the carotid of a horse, immediately above the giving off of a large branch is a point of great value, (see experiment 8); for if the permanent safety of torsion depended upon the mere

coagulation of the blood in the vessel, success could not have been thus acquired—for as the drawing, model, and preparation well show, there was no room for such an obstruction to the arterial flow to take place between the twist in the cellular coat, and the point at which the branch artery was given off.

It would thus appear that the twist in the cellular coat was the chief cause of the arrest of the arterial hæmorrhage in the experiment referred to, and that this twist with the adhesion of the split, *retracted*, and approximated middle tunic forms the true permanent occluding medium of a divided vessel; that upon such points the main safety of the practice of torsion really rests, the coagulation of the blood in the vessel acting a part of secondary importance.

As mere experiments, these operations on the horse must be looked upon with favour; for they prove practically that torsion of such a large artery as the carotid is to be relied on; and consequently it does not appear unfair to believe that the same practice applied to any smaller vessel would be equally successful. As physiological experiments, however, the observations I have been enabled to make must be regarded in a higher light, for they show that nature's well-known hæmostatic processes act at a far greater advantage when torsion has been applied, than they do after the use of the ligature; and, what is of far greater importance, they are allowed to go on undisturbed in their conservative action, for there is no foreign body to set up ulcerative action in the vessel in the seat of its occlusion; nothing to undo at a late period of the case what nature, assisted by art, has done at the beginning for the prevention of arterial hæmorrhage. The twist in the vessel remains permanent; the coagulation of the blood in the meshes of the retracted, approximated and incurved middle coat goes on undisturbed; and the whole becomes, subsequently, rapidly condensed into one homogeneous mass by the effusion of lymph from the exposed surfaces of the divided tunics.

In the application of torsion some care is needed, and as two methods have been described, the "free" and the

"limited," it may be well to point out as far as possible the conditions under which they should be employed.

"Free" torsion appears to be the more applicable to small vessels, and even to the large trunks of the extremities; the "limited" torsion to such arteries as are unsupported by muscles and loosely connected.

It will have been observed that in all my experiments upon dogs, in which both carotid and femoral arteries were treated by torsion, the "free method" was used; and in the single example I have quoted in which I adopted the practice in a large vessel on the human subject—amputation of the arm high up—free torsion was also employed; for in that case I passed one blade of my forceps into the humeral vessel for about half an inch, and, with the other outside, after four complete revolutions the operation was successfully performed. I have, since this paper was written, applied free torsion to the femoral artery of three adult patients with complete success. On the dead subject also this mode of torsion proves successful, particularly on the femoral, brachial, and carotid arteries. It should also be stated that Amussat in his last years employed the *limited* torsion in all except the very small vessels; whilst Thierry employed the *free* in every case.

Three or four revolutions of the artery are required for small arteries, five or six for large: when the vessels are atheromatous the former number (three or four) is amply sufficient under all circumstances.

The orifice of the artery alone should be held by the forceps which twist the vessel in both cases.

In no case does it appear necessary, or even prudent, to twist the vessel till the end is twisted off: for by so doing we do away with the chief safeguard against bleeding, we run the risk of breaking up the valvular incurvation of the inner tunics, and disturb the clot which forms between the retracted ends of the two inner tunics and the twisted cellular coat. In very small vessels this point is not of so much importance. It is probably due to this error of twisting off the ends of the vessels, that the practice of torsion has failed to hold its ground, for in the few cases in which

it has been employed, such a mode has been generally, if not universally, adopted.

It seems to be a good rule in every case of torsion, whether free or limited, to hold the end of the vessel for a second or two after the twists have been made, to allow of the parts becoming plugged with blood, and the divided tunics to retract and come in contact.

It appears also to be a matter of great importance in twisting large vessels that the whole orifice of the artery should be seized and firmly held; this cannot be well done with the forceps ordinarily sold, but can be by the forceps I have had made for the purpose by Mr. Millikin of Southwark Street, the ends of which are broad, and firmly hold the artery, and not having sharp ends do not tear it. The blades are closed by a simple catch. With a small pair of forceps the surgeon is very apt to tear out a piece of the end of the vessel and not twist it securely. I have proved this on many occasions on the dead body.

The forceps I have had made for holding the vessel are very simple, they fix it firmly with flat surfaces without injuring the external coat, which the sharply-teethed forceps are apt to do; they are also very well adapted for the twisting of vessels of moderate calibre.

Comparison between Torsion and the Ligature.

If we compare the effects of torsion on the arteries with those of the ligature, it appears that the changes the two inner coats of the artery undergo are in a measure similar under both circumstances; that the same plugging of the vessel by the temporary clot, and permanent sealing of the extremity of the vessel by the organization of effused lymph, take place in both—in the one case the twisted cellular or external coat mechanically arrests immediate hæmorrhage, and in the other the ligature. But between the two methods there is this great difference, that with the ligature we have a foreign body, applied only for a temporary purpose, which,

when it has served, can only be regarded as a source of irritation and of some danger; but with the torsion no such foreign body exists, for, when the extremity of the vessel has been once fairly twisted, it is twisted for ever, and what was at first only a temporary obstruction to the arterial flow becomes a permanent one.

In the second experiment on the horse which has been modelled (see drawing from model, Plate IX), all these changes may be seen, and from this it would, moreover, appear that the twisting of the vessel, together with the adhesion of the orifice of the two inner tunics, is amply sufficient of itself to be both a temporary and permanent obstruction to the arterial flow; the formation of a clot being of quite a secondary importance.

It should be stated, however, in this comparison that in the ligature there is no incurvation of the ruptured middle or inner tunics, and but little retraction. It would thus appear that when torsion has been thoroughly and efficiently performed, hæmorrhage from the small, and even from the largest vessels, may be arrested with some certainty; and that, what is of still greater importance, when this effect has been produced, the fear of secondary or any subsequent bleeding is apparently groundless: for when once the inner tunics of the arterial walls have been divided, and as a consequence have retracted, incurved, and approximated, and when the cellular coat has been completely twisted upon itself more than once, the knot formed by such a twist does not seem to be capable of unfolding, and there is no foreign body about the vessel, as there is when a ligature has been used, to set up a fresh inflammatory or ulcerating process to destroy the permanent sealing of the twisted or occluded artery, and thus give rise to secondary hæmorrhage.

It is in this point, I would respectfully suggest, lies one of the great advantages of torsion over the ligature, for when once bleeding has been successfully arrested by well-applied torsion, and no signs of hæmorrhage occur within a few seconds of its application, there need be little fear of a subsequent bleeding—in a healthy subject at least—or of a

secondary hæmorrhage, for there is nothing to interfere with the physiological processes which nature invariably adopts to seal permanently the divided vessel; there is no foreign body to set up ulcerative action, which must undo to a degree, and may undo altogether, the work that nature has performed to guard against bleeding.

In the use of the ligature no such certainty exists, for such ulcerative action must take place for the purpose of its liberation, and this ulceration, or sloughing of the ligatured extremity of a divided artery, is the most common cause of secondary hæmorrhage; we all know, also, too well how frequent and how serious secondary hæmorrhage from a large artery always is.

I might add that of all the cases recorded, in which torsion has been used, in none is secondary hæmorrhage mentioned to have taken place.

It may be objected, however, that torsion is not applicable in every case. I would perhaps grant the objection; but because a practice is not applicable in every case, there can be no objection to its use in such as it can be applied. I believe it will be found that the practice can be carried out in by far the majority of cases of divided vessels, if not in all, whether healthy or diseased; but that in the latter the number of twists required will be far smaller than in the healthy subject, for when arteries are diseased it is in the middle and inner tunics that the mischief is situated, and it will be found that these coats readily break under the influence of torsion, and as readily retract and approximate; the cellular coat is rarely involved in the disease, and may be twisted with care.

Again, should torsion fail it will fail at once, and the surgeon can then repeat the same or adopt other means of arresting bleeding; but should it succeed, it will probably succeed unto the end, for there is no local exciting cause to set up fresh action, there is no influence going on in the part to undo at a later period of the case what has sufficed to arrest the flow of blood at the beginning.

By way of summary the following conclusions may be drawn up.

1. That hæmorrhage may be arrested by torsion from even the largest vessels.

2. That it is a safe and judicious practice in all cases in which the vessels are small or of moderate calibre; and that, as far as experiments and practice yet prove, it is equally so in arteries of the first magnitude.

3. That torsion may be "free" or "limited:" the *free* method being applicable to vessels of moderate size, and even to the largest of the extremities; *limited* torsion being more adapted for the large and loosely connected vessels.

4. That in *torsion* as in the *ligature* the permanent hæmostatic processes are alike due to the sealing of the divided inner and middle tunics; but that in the ligature there is only an irregular division of these tunics, whilst in torsion there is a complete division, separation, retraction, and valvular incurvation.

5. That in torsion the *twisted cellular* coat forms, with the retracted and incurved middle coat, the direct mechanical obstacle to the flow of arterial blood, in the same way as the *compressed cellular* coat does in the ligature; but that in torsion the twisted cellular coat and incurved middle coat become subsequently a permanent means of occluding the end of the artery; whilst the ligature of necessity becomes subsequently a source of irritation, and too often a means of undoing what has been done by nature's own hæmostatic processes.

6. That in *torsion* the twist in the cellular coat of an artery; the division and subsequent retraction, incurvation, and adhesion of the middle coat; and the coagulation of the blood in the vessel down to the first branch, are the three points upon which its temporary as well as permanent safety depends; whilst the permanent safety of *acupressure* rests only upon the last point alone, and its temporary effects upon the pressure produced by the needle.

7. That there is every reason to believe that when torsion has been successful on its first application, the fear of subsequent hæmorrhage is altogether groundless; for there is nothing, as there is in the ligature, to interfere with the physiological processes set up by nature to occlude the divided vessel; and, unlike acupressure, the temporary obstacle to the flow of blood becomes a permanent one.

8. That upon physiological grounds torsion has decided advantages over the ligature and the acupressure needle; and that if subsequent experience confirms what has been hitherto observed in the experiments on animals and the application of the practice in the human subject, we shall have gained a point of no mean importance and simplified surgery in no slight degree.

Conclusion.

I have thus, as tersely as I could, brought forward all the facts of which I am cognisant bearing upon torsion as a hæmostatic agent, and some of the arguments by which it may be commended to your consideration. I trust I have adduced sufficient to prove it to be well worthy of extended trial.

That it is a reliable practice in many cases when efficiently performed can hardly be disputed; in how many, can only be proved by the ultimate test of all knowledge—experience. It is not a crude idea based upon a theory spun out of a fertile imagination; for it had its origin in observation of nature's own processes; it is based on the well-recognised physiological principles of natural hæmostatics; and it is artificial only so far as the surgeon's art is employed in rendering these processes most available.

Nearly forty years have now passed since its distinguished originator brought it before the notice of our foreign brethren; but I trust before another decade has passed it will have been tested to the full, and that when weighed in the balance of experience it will not be found wanting, for I entertain a high opinion of its capabilities, and believe that in the future it is destined to be a handmaid to the surgeon of no secondary importance.

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DESCRIPTION OF PLATE VIII.

Fig. 1.—Brachial artery from a case of amputation, in which hæmorrhage was arrested by torsion (see pages 207–9). Mode of preparation—artery slit by scissors up to dot; then hardened in spirit, and a longitudinal section made with a sharp knife. Drawn to natural size.

A. Tunica adventitia; at A′ twisting.

B. Tunica media and tunica interna, without visible line of distinction.

B′. The same, at point of torsion, ruptured and retracted towards the cardiac end of the artery, at B″ towards distal end, but not so perfectly incurved.

C. Clot. C′ between tunica media and adventitia at point of torsion. C″ in part of artery beyond point of torsion.

D. Small artery coming off above dot.

*** The arrow shows the direction of the blood current.

Fig. 2.—Carotid artery of a dog, showing the effects of torsion (magnified one third). Experiment No. 4. (See pages 210–11.)

A. Brachio-cephalic artery, dividing into subclavian and carotid.

B. Part of artery distal to the cut.

*** The arrows show the direction of the normal flow of blood.

d, *d*. Middle and internal coats; at *d′ d′* very little incurvation; at *d″* much thicker than elsewhere by contraction.

e, *e*. External coat; at *e′* twisted.

f, *f*. Clot, here and there decolorized.

g. Ampulla.

h, *h*. Portions of artery seized with forceps, much shrunken.

Fig. 3.—Effects of torsion on the living carotid artery of a horse. Experiment No. 7. (See pages 212–13.) Mode of preparation—hardened in spirit and a longitudinal section then cut. Drawn to natural size.

A. Portion of artery on the cardiac side of the cut.

B. Distal portion.

C, C. Corresponding ends of the artery where it was cut and twisted.

*** The arrows represent the normal direction of the current of blood before the artery was touched.

d, *d*. Middle and internal coats turned in so as to meet at *d′*, with a kind of knob-head at *d″*, the same merely overlapping each other.

e, *e*. Internal coat; at *e′* separated from the middle by a small quantity of decolorized clot; at *e″ e″* points of twisting.

g, *g*. Portions of artery seized by the forceps: these show no appearance of sloughing, but rather of atrophy.

h, *h*. Clot, in spots decolorized; from *h′ h′* a piece which had contracted no adhesion to the arterial walls, but which fell out in making the section.

x. Pouch-like swelling containing decolorized clot, and elastic lamellæ from middle coat of artery (?).

Fig 1

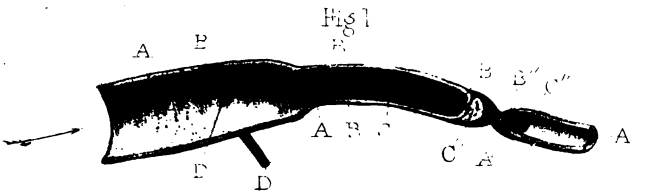


Fig 2.

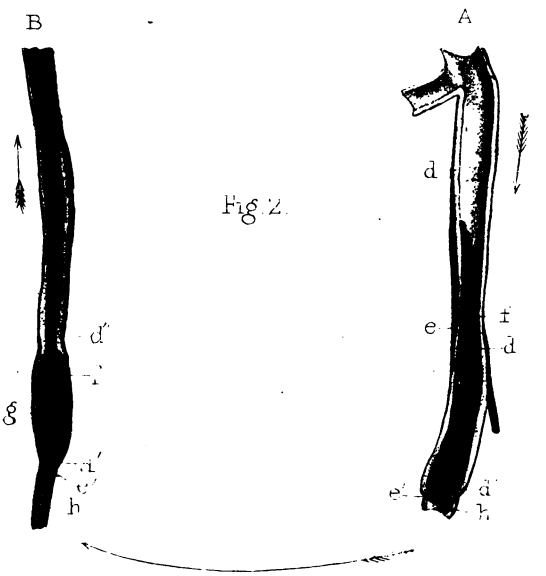
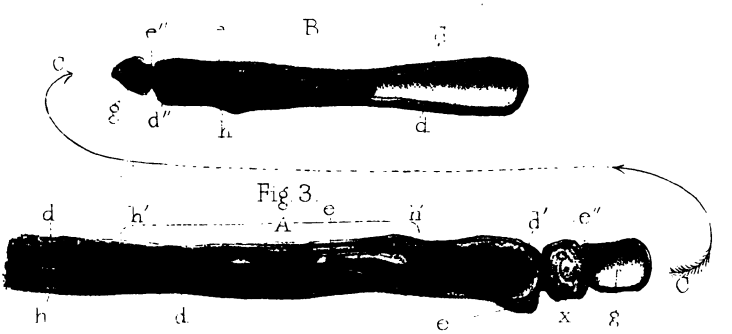


Fig 3.



DESCRIPTION OF PLATES IX AND X.

PLATE IX.

Illustrating Experiment No. 8 on the horse. Taken from Mr. Towne's model.

*** The arrow indicates the course of the blood. (See pages 213–14.)

Fig. 1.—Carotid artery of a horse to which torsion had been applied.

A. The external coat which had been twisted.
B. The retracted and adherent middle coat.
C. Clot in the meshes of the split middle coat.
D. Large branch which was open.

Fig. 2.—Carotid artery of a horse to which a ligature had been applied.

A. Ligature which had nearly ulcerated through the vessel.
B. Divided and adherent middle and inner tunics.
C. Clot in the free extremity of the artery which has contracted upon it.

PLATE X.

Figs. 1, 2, 3.—Arteries laid open to show the valvular incurvation and splitting of their middle and inner coats the result of torsion (see page 215).

A. Twisted external coat.
B. Valvular incurvation of middle and inner coats.
C. Splitting of middle coat.

Fig 2

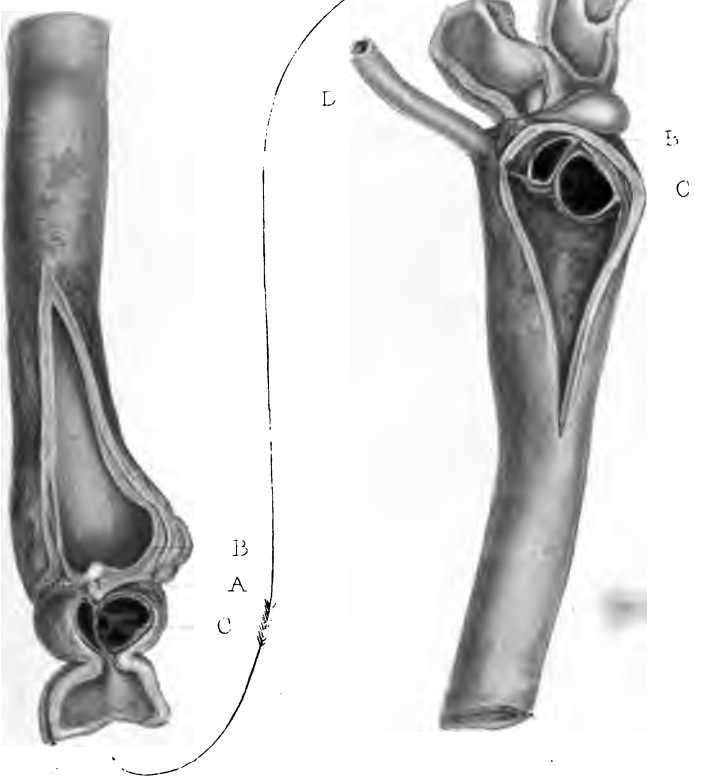


Fig 1

Fig. 1.

Fig. 2.

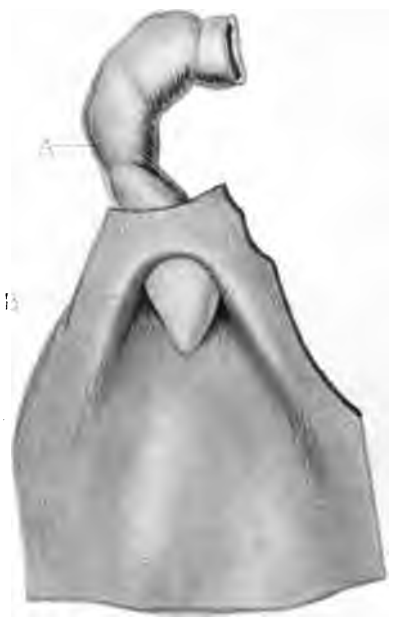


Fig. 3.

CASE OF

PERFORATION OF THE MASTOID CELLS:

SUBSEQUENT FORMATION OF AN ABSCESS BENEATH THE STERNO-MASTOID MUSCLE.

BY

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Received April 27th.—Read June 23rd, 1868.

B. J—, æt. 58, a clergyman, of healthy constitution, applied to me on the 22nd of February, 1867, on account of a discharge from the left ear. A month before, after officiating in a cold and draughty church, he was seized with pain in the ear, which continued severe for eight days, when a discharge appeared. The relief that followed was, however, of short duration, and pain and discharge had since continued unabated. There had been no previous disease of the ear, except a slight and temporary deafness some years before, after influenza.

On examination there was found an abscess in the left tragus, which nearly closed the ear. This was opened, and the meatus was found to be much swollen, and its surface desquamating. The membrana tympani was red, and presented an irregular orifice in the lower part, about a line and a half in diameter, through which air passed freely when the patient inflated the tympanum. During the three days he remained in London, the ear was freely washed with a weak

solution of sulphate of zinc and morphia, and a large quantity of discharge removed from the tympanic cavity. On his returning to the country the same lotion was directed to be used twice daily; air being at the same time passed through the tympanum, and the ointment of iodide of potassium with extract of aconite to be rubbed around the ear. No relief, however, followed. Severe attacks of pain recurred regularly about 3 a.m. For these quinine was given (two grains every four hours), and the pain diminished for a time but soon returned, severe and constant, over the vertex and back of the head. Some tenderness also was at one time found about an inch behind the meatus, but this passed off.

On March 12th I again saw the patient. There was then no swelling or tenderness over the mastoid process. Very large quantities of tenacious secretion were removed by syringing, from the tympanum, its exit being aided by the use of sulphate of zinc, and the passage of air through the Eustachian tube. The meatus was more healthy. The orifice in the membrana tympani was larger, and the exposed mucous membrane of the tympanum appeared red and irritable. The membrana tympani had lost its redness, and was of a dull white hue. A physician was consulted, who advised change of air and prescribed a mercurial purgative with sulphate of bebeerine. A lotion of borax and opium was advised.

In the course of a week the patient returned again from the country, with an increase of pain; and a slight degree of swelling and tenderness were found over the mastoid process. The discharge continued profuse; not very fetid. An incision through the periosteum of the mastoid process was now advised, but owing to the great reluctance of the patient it was deferred, and on the next day, under the use of fomentations, the swelling and tenderness had almost entirely disappeared. A succession of small blisters was advised, and for a few days the patient appeared to improve. On the 30th of March, however, I received a letter from his wife, stating that the pain had greatly increased, especially at the

vertex, and that there was an increase of the swelling around the ear, with general feeling of illness. Under these circumstances I visited the patient, and found that indications of disease affecting the mastoid process had assumed a decided character. The part was much swollen, red and tender; there was constant severe pain at the top of the head, greatly aggravated by coughing or sneezing. Pulse 100; frequent cold perspirations.

Without further delay an incision one inch and a half in length was made through the periosteum, covering the mastoid process, about half an inch behind the ear. The soft tissues were swollen to upwards of an inch in thickness. Considerable relief followed, but not complete; and on examination the next day I found that there was a slight roughness and softening of the bone, about the central and lower part of the process. Through the most softened part I introduced a medium-sized trocar, boring without much difficulty to the depth of a little more than half an inch. On withdrawing the instrument a free stream of dark grumous pus followed; not offensive. The opening was enlarged and more pus escaped. The wound was kept open by oiled cotton.

The pain was at once almost perfectly relieved. In twenty-four hours the discharge from the meatus entirely ceased, and on the third day after the perforation of the bone, the orifice in the membrane was healed; air blown into the tympanum no longer escaping into the meatus. Bare and softened bone was felt at the bottom of the wound. On the tenth day no more bare bone was to be felt, and the wound was healing. Membrana tympani of a dull pinkish hue; in the position of the orifice was a thick white patch. Air entered the tympanum with a slight creaking sound. There was no swelling, but a slight pain in the side of the head, at about 4 p.m. daily; this, however, soon subsided, and in a few days more there remained only a small sinus, at the bottom of which no bare bone could be detected. The hearing also had very considerably improved.

At this time (about the middle of April) the patient spent

a month at Torquay, during which, after some exposure, there was a return of pain, intermittingly, in the ear and head, which did not entirely subside. But on examination, on his return to town, no sign of local mischief was detected, and he went into Norfolk. There the attacks of pain increased, especially at the vertex, and some shiverings occurred, after which a large firm swelling formed in the side of the neck, bounded above by the mastoid process, and extending downwards and forwards for about four inches in the course of the sterno-mastoid muscle. Four leeches had been applied without relief. There was much febrile disturbance, and frequent and profuse perspirations. No fluctuation was discernible. A slight oozing of pus, however, had continued from the small sinus which existed in the position of the incision previously made, and from this sinus a little dark matter could be squeezed on pressing the swelling upwards.

May 24th.—The former incision was extended downwards about half an inch, the knife being carried deeply into the softened bone, so as to lay the mastoid cells freely open. Free bleeding took place, and blood also escaped into the pharynx, evidently passing through the Eustachian tube. The bleeding was controlled by pressure on the anterior part of the mastoid process. A blunt probe passed freely into the bone for the distance of about an inch and a half, and gave exit to some red-coloured matter, and in the course of the day about a dram oozed out with some relief to the pain. The bulk of the swelling, however, remained entirely unaffected.

26th.—Pain less; a slight escape of purulent matter, not fetid, on the poultices. Swelling in the neck undiminished; a puffiness extending anteriorly over the region of the carotid.

A very small trocar and canula were introduced at the most prominent part of the swelling, about two inches below the mastoid process, and through the substance of the sterno-mastoid muscle. At the depth of about an inch and a half a drop or two of reddish matter escaped on withdrawing the trocar, and on introducing a larger canula, nearly half an

ounce was evacuated. The canula was left in the wound, and spongio-piline applied. Pus continued to flow, and the pain in the head became much less. It was distinctly aggravated, especially at the vertex, whenever the discharge of matter ceased. On the fifth day free bleeding took place from the wound, which, however, was stayed by pressure before many ounces had been lost, and did not return.

On the 8th day Mr. Cadge of Norwich saw the patient in consultation: wound kept open by lint. Swelling less.

Fifteenth day (June 9th). Swelling diminished and more circumscribed. Pains in the head not less severe, but always relieved by the escape of matter. A curved probe, introduced through the wound, after passing inwards about an inch and a half, could be turned in all directions, evidently in the cavity of an abscess; most freely downwards. A slight discharge escaped now and then from the superior opening, but no connection could be traced between it and the abscess.

Perforated laminaria tents were introduced, which were very effective in giving free exit to the matter, but were too painful to be continued.

The condition being evidently an abscess of considerable size beneath the sterno-mastoid muscle, and probably within its sheath, it was considered necessary to have recourse to further procedures, and on 12th June the patient was removed to London. He was here seen by Mr. Paget, and on the next day, by the advice and with the kind assistance of that gentleman and Mr. Cadge, the abscess was laid freely open in its whole extent by means of a curved bistoury and director; the incision being made continuous with that originally made over and into the mastoid process. The bleeding from the divided occipital artery was controlled by plugging. No dead bone was exposed by this incision, nor was any subsequently detected; although for a time a probe could be passed for nearly two inches into a small sinus-like canal at the bottom of the wound. This, however, soon closed, and the wound healed in about two months. In September, three months after the opening of the abscess, and six

months after the perforation of the mastoid process, the patient enjoyed his customary amusement of shooting.

March, 1868.—The orifice in the membrana tympani continues soundly healed, showing a small scar; the hearing of the affected ear is but little impaired, ordinary conversation being heard with ease, and a low watch at a distance of six inches.

In respect to the formation of the abscess beneath the sterno-mastoid muscle, consequent on inflammation within the mastoid cells, this case stands, so far as I am aware, by itself. It is, however, only one of several which seem to establish the benefit of perforation of the mastoid process, if performed sufficiently early. Of such cases I have met with an account of fourteen, in twelve of which the operation gave perfect relief, while in two fatal mischief already existed within the skull. Of these, eight, all successful, one being his own, are collected by Dr. Von Troeltsch in Virchow's 'Archiv' for 1861:—three, of which two were successful, are reported by Dr. Pagenstecher in the 'Archiv für Klinische Chirurgie' for 1863;—one, successful, by Dr. Schwartze in his 'Praktische Beiträge Zur Ohrenheilkunde,' p. 37;—one, also successful, by Dr. Flaiz in the 'Archiv für Ohrenheilkunde,' vol. i, and one, in which relief was given, but pyæmia had already set in and proved fatal after some weeks, by Dr. Mayer in the same 'Journal,' vol. ii. Of these cases the one related by Dr. Flaiz presents many points of similarity to my own. It is briefly as follows:

A clergyman, æt. 46, in good health, was attacked with inflammation of the left meatus and tympanum. In a few months fever set in, with twitchings of the left arm and shoulder. The membrana tympani was perforated, air passing through it on inflation. Swelling and tenderness over the mastoid process. After leeching, without relief, an incision was made through the periosteum, evacuating half an ounce of pus; the bone was not rough. The pain ceased, but as the wound healed it returned with great severity. Delirium came on at night, and the left arm was in constant motion. There were two especially painful spots; one at the occiput the other on the left parietal protuberance.

The incision was reopened, and a perforator introduced on a level with the external meatus. The cellular structure was reached at a depth of three lines, the instrument being carried somewhat forward and upward. The opening was then enlarged by a probe; but no matter appeared until after thirty-six hours, when an abundant discharge ensued and lasted for some days. In four weeks the patient resumed his duties, free from all symptoms, and contented with his hearing.

The indications for the perforation of the mastoid process are essentially the same as those now generally recognised as demanding an incision through the periosteum covering it, with the addition that entire relief is not given by the latter procedure. The symptoms are severe and continued; there is pain in the ear or head, especially on moving, which is accompanied or preceded by a discharge from the ear, and by tenderness on pressure over the mastoid process. If, in addition, symptoms of cerebral irritation are present, or if the bone on exposure is found bulging or softened in certain parts, the necessity for perforation is of course more evident.

I omit from the list of indications swelling and redness over the mastoid process, for although these symptoms are generally present, yet it appears that they are sometimes absent when perforation of the bone affords the only prospect of saving life: in those cases, for example in which necrosis extends from the meatus or tympanum, not downwards so as to involve the mastoid cells, but inwards into the upper portion of the pyramid. In such cases there may be little or no swelling externally, and yet an opening through the upper portion of the mastoid process, about on a level with the roof of the meatus externus, may enable the necrosed portion to escape, or at least may give a safe outlet to the fluid that surrounds it.

A case of this kind, but in which the attempt was made too late, is reported by Dr. Pagenstecher ('Archiv für Klinische Chirurgie,' vol. iv, p. 529).

It is scarcely necessary to observe that shortly after its

successful performance by Jasser in 1776, perforation of the mastoid process was frequently resorted to as a remedy for deafness, until the death of Dr. Burger, in 1791, led to the total abandonment of the proceeding. It would of course never be proposed now except to avert a pressing danger.

In conclusion, I may briefly relate another case, very similar in some respects to the preceding, but in which perforation of the mastoid process was not performed:—

October 12th, 1866.—B. T—, æt. 60, a publican, but of moderate habits, was seen by me with Mr. Geo. Roper. Three months before he had suffered from severe pain and discharge of pus from the right ear, which have continued since in a less degree. For the last week the pain has again been more severe. He had been subject for two or three years to pain and swelling in various joints, but these have been absent since the ear became inflamed. The hearing was almost abolished on the right side; the mastoid region was slightly swollen and tender, but not red. The posterior wall of the meatus, just in front of the membrana tympani, was red and projecting so as nearly to fill the passage, and from amongst some granulations on this surface pus was seen to issue. No dead bone, however, could be felt. Air could not be passed into the tympanum, and the use of the Eustachian catheter was refused. No fever; tongue clean. Leeches were applied; and an incision over the mastoid process advised, but the patient would not consent until the end of a week, when all the symptoms had become aggravated. An incision was then made, but no dead bone was found, nor did any pus escape from the wound, though on pressure a large quantity flowed from the meatus. Air was passed through an orifice in the membrane.

Great relief was given to the pain; but the patient became weaker, and in the course of a fortnight a large abscess had formed in the pharynx on the right side. This was opened (also after delay on the patient's part) by Mr. Roper, and discharged freely, as also did the wound behind the ear. Still no dead bone was felt. On November 19th, four months

after the commencement of the attack, he died with symptoms of pyæmia.

The post-mortem examination was conducted by Mr. Roper, to whom I am indebted for the temporal bone, and for the following brief report.

The substance of the brain seemed unaffected, and the membranes also, except a patch of ecchymosis on the basilar process of the right sphenoid bone; but the bone itself was not involved. The parts beneath the base of the skull were infiltrated with pus, and formed to a large extent the sac of an abscess, which was bounded superiorly by the base of the right petrous bone. There were also other abscesses in various parts of the body, the largest of which was situated over the right scapula.

Right temporal bone.—The mastoid process was reduced to a mere shell, breaking down under slight pressure, but not carious externally. The internal portion formed a cavity in which lay a carious fragment of bone, about the size of a large bean, and evidently consisting of the remains of the cellular structure. The jugular sinus was obliterated, and the portion of the vein attached to the bone contained a sanious mass. The dura mater at this part was thickened and white, but not ulcerated. The walls of the tympanum did not appear to be carious, and the bony meatus seemed healthy. The membrana tympani was soft and swollen, and presented a small perforation at the posterior and superior part.

On looking at the preparation I think it is hard to resist the conclusion that an external opening into the mastoid cells might have saved the patient's life. But it seems to me also worth consideration whether (considering the small size of the orifice in the membrane, which was scarcely discoverable in the recent state, and in the dry membrane does not equal a line in its greatest length) a free enlargement of this orifice, so as to allow an easy escape of fluids from the tympanum, on the first access of symptoms of extended irritation, might not have been even more efficacious.

ON

PARALYSIS OF THE EXTENSORS.

BY

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Received June 9th.—Read June 23rd, 1868.

THE object of the present paper is to illustrate by the few cases that have come under observation a rare form of paralysis, which is sometimes confounded with diseases of a totally different nature, and hitherto, as far as the writer is aware, has escaped description.

Until recently no opportunity was afforded by a fatal case of ascertaining the morbid alterations in this interesting form of disease, and as diagnosis was consequently verified by post-mortem examination in only one instance it seemed advisable to confine the remarks to be made to a simple description of the cases under notice, trusting to future observations for a more accurate portrait of the disease.

In the form of paralysis here illustrated the extensors of the hands and feet are paralysed, either or both forms of paralysis being present, and a patient is easily recognised by the dropped hands and analogous condition of the feet. This condition may be preceded by hyperæsthesia, or associated with some altered sensation which will serve to distinguish this form of disease from lead poisoning. In none of the cases observed was any history of lead contamination elicited, nor was there any evidence of the presence of lead by the blue line round the gums.

On looking over the post-mortem registers of St. George's Hospital, which have been carefully kept since 1845, I have only been able to find one instance of this disease, recorded by Dr. H. W. Fuller, in 1846. The accurate description there given leaves little doubt in my mind that the case was similar to those that are here recorded, and though unfortunately the spinal cord was not examined, the case is quoted as entered in the register.

The first case that attracted notice was that of a man, æt. 41, admitted March 28, 1866. He was a pork butcher, a large corpulent man of full habit. He had been accustomed to live freely. The history he gave was that he had suffered from pains in the limbs attributed to rheumatism, two months before admission. Two weeks before admission he was suffering from decided pain in the knees and feet, which was followed by dropping of the hands. There was no history of lead poisoning, and no blue line on the gums. On admission he had all the appearance of a free liver: very stout; face congested and dusky; no evidence of gout, nor of rheumatism; the urine was not albuminous. He complained of aching pain in the hands and feet; there was paralysis of the extensors of the hands, which were flexed but lax, and loss of power in the feet. It was not noticed in this case that the feet were affected in the same way as the hands. He was dull and heavy, but he answered questions intelligently, and there was no symptom to lead to the conclusion that the brain was diseased.

He was ordered to take ʒj of the Liq. Hyd. Bichlor. three times a day, which seemed to produce some improvement of the general state of health. But the paralysis was progressive, and the pains gave place to numbness affecting the lower extremities. Subsequently strychnia was injected subcutaneously, but this caused so much tingling that the patient refused to submit to it again, and he was ultimately sent out incurable.

A woman, æt. 46, was admitted in May, 1866, with a

history that she had been suffering from paralysis of the legs and feet, which had come on suddenly four weeks before admission. It was ascertained that she had been lately drinking very freely; she was a very corpulent woman of full habit. She was quite helpless, could not walk nor feed herself, and on admission there was paralysis of the extensors of both hands; the feet were also affected, but it was not noticed that the extensors only were affected. No history of lead poisoning, and no signs of lead in the gums. She complained of pains in the head and about the legs, and there was numbness of the upper extremities. She was extremely low spirited. She was treated with the biniodide of mercury, strychnia injections, and the tincture of nux vomica, and she improved slightly, so far indeed as to feed herself, though with much difficulty. She was four months in the hospital, and dismissed as incurable.

A woman, æt. 38, was admitted into St. George's Hospital on the 9th October, 1867, with the following history and symptoms. She had enjoyed good health until attacked with pains three months before admission. These pains were attributed to rheumatism. There was no evidence of either rheumatism or gout. She confessed to having been a tippler of ale and gin, and for some time had slept but little. When seen she was apparently on the verge of delirium tremens; was very nervous, tremulous, and jocular in manner: the face was dusky, the facial veins much congested; the urine was carefully examined, but found natural; the tongue was clean. She was not in a state to give any accurate account of herself; but her speech was natural and her answers coherent. She was delirious at night, and was very noisy, requiring large doses of opium to keep her quiet. After she had been in the hospital three weeks, it was observed that she was losing the power of grasping; she was very sensitive and would cry out hysterically when touched, subsequently both hands became dropped; they were perfectly lax, and there was no contraction of any of the muscles; the hands fell by their weight in the form of extension or flexion, in

whatever way they were placed, and they were quite useless to perform any of the ordinary actions. The prominent parts of the palms were very red, and erythematous; there was a little more power in the right hand than the left; there was well-marked hyperæsthesia of both upper extremities.

She could at this time move her legs, but was very helpless, and lay in bed motionless and taking very little interest in anything about her unless roused, when she would talk jocularly and laugh in an excited manner.

No history of lead poisoning was obtained in this case, and there was no evidence of lead about the gums.

A few days afterwards the feet were observed to be affected in a similar way as the hands. The feet were turned inwards and the soles slightly upwards, and there was no power of motion in the feet. The same hyperæsthesia and the same erythematous blush were observed here as in the hands. Galvanism was applied and found to produce considerable pain, but no increase of power. She continued in the hospital for nearly four months. She became weaker, and more helpless, and much thinner, and was finally sent out as incurable. When she went out the hands and feet were in the condition above described, there was slight wasting of the interosseous muscles, the hyperæsthetic condition being not so marked. She had no power of moving the left leg, and but little of moving the right. The urine was not albuminous; the pulse was 80 in the minute.

A woman, æt. 32, was admitted October 13, with symptoms of paraplegia. The history she gave was that for the last three months she had been drinking freely, having been much distressed at the loss of a child. For seven weeks she had been getting weak in her legs. On admission there was no diminution of sensibility in the legs, but there was some loss of power; the feet were turned in and the soles upwards; there was some redness of the soles; the left foot was more affected than the right; there was a condition of general

weakness, and her manner was excited, her face rather congested and dusky. She passed her water under her. Subsequently she complained of weakness of the hands, but no dropping of the wrists was observed. No lead was observed about the gums. She improved with strychnia and galvanism sufficiently to walk without help, but the feet were a good deal turned in and the sole was put down quite flat; no albumen was found in the urine.

A young girl was admitted on April 18, 1868, with the following history. She was a prostitute (æt. 19), accustomed to drink a great deal, and her companion mentioned that she had been used to take considerable quantities of spirits at a draught. The history the patient gave of her illness was, that seven months before admission she caught cold, and this was followed by loss of power in the hands, which shook when she tried to lift anything. In three weeks she lost all power in the hands, and there was also some loss of power in the legs, but she was able to walk even up to the 9th of April. One month before admission she had two fits of an epileptic character. When she was admitted her hands were dropped and useless; her face was full and dusky, there was nothing hysterical in her manner, but she had the appearance of a debauchee; there was complete sensibility of the hands and no loss of heat; the feet were useless, the soles being turned inwards; there was slight loss of feeling in them, pulse 100. Tongue slightly furred; urine not albuminous. The catamenia had ceased altogether for seven months.

On the 16th she was observed to become very irritable and cross in the evening, and at 1 a.m. on the 16th she had a fit of convulsions lasting four hours, after which she lay in a comatose state, rolling her head on the pillow; breathing stertorous; pupils both equal and small. She moved the left leg when the soles were tickled, and there was very slight reflex action in the right foot. The left arm was apparently quite paralysed, but she moved the right arm. She subsequently had another fit on the 26th, in which she died.

At the post-mortem examination, which was made by Mr. Pick, the following notes were taken:

The examination was made 20½ hours after death.

The body was natural in appearance.

The lungs were œdematous, the bronchi congested.

The left side of the heart was contracted and empty, the right dilated and full; the valves and structure were natural.

All the abdominal viscera were perfectly healthy.

There were a number of soft condylomatous growths just within the vagina.

No tubercle was detected, though carefully looked for.

The brain throughout was very pale, and was also rather wet, a considerable amount of serum dripping from it on section, but with this exception it was quite natural.

On exposing the spinal cord it was found to be softened in two places; the upper lesion was opposite the sixth cervical, the lower opposite the twelfth dorsal. These lesions were rounded and formed a limited swelling (the size of a threepenny piece $\frac{4}{8}$ inch $\times$ $\frac{3}{8}$) on the posterior columns with a depression in the centre of the swelling, the seat of considerable softening. The upper portion in the cervical region was laid open, the lower was put aside and hardened in chromic acid. The spinal cord, elsewhere, seemed to be in its natural condition.

The extensor and flexor muscles of the forearm were examined, and were found to be in a very early stage of fatty degeneration; the muscular substance was rather paler than natural, and less striated than in a healthy condition, but no fat-globules were found in the tissue, and it was quite clear that the disease was not primary in the muscles.

The musculo-spiral and the median nerves were examined, and the former was found to be considerably attenuated and much smaller than the median, which was of normal size. The peroneal nerve was also found much diminished in size.

The following case was admitted on July 22, 1846, under Dr. Wilson: the notes of which are due to Dr. Fuller. The patient, a woman, æt. 40, had been ailing for two years,

during which time she had felt weak and languid, and had suffered from pain in the head. Nothing more than these general symptoms was observed until eight or nine months ago, when she began to lose power over the extensors of the hands, and the general symptoms of dyspepsia were somewhat aggravated. She continued to work up to eight weeks ago, when a bilious attack supervened. The palsy of the extensors of the hands became more complete. The general symptoms since that time have been much more severe, so much so indeed that she had hardly been a day without vomiting.

On admission her complexion and the conjunctivæ were somewhat yellow. She had dropped hands as if from lead poison, but no history of the kind could be obtained, and there was no blue line along the gums. The right hand was rather more affected than the left, which in the first instance was the worst of the two, but had partially recovered after being bandaged up in tea lead. Her tongue was clean but fissured, urine abundant, acid, sp. gr. 1008, albuminous. She suffered from constant vomiting. On the 25th she suffered from diarrhœa; the bowels continued relaxed until 3rd of August, when in the afternoon she had a violent fit of an epileptic character which left her in a comatose state, and apparently moribund. Eight fits of the same character occurred in the course of the day.

The next day, after having passed a very restless night, talking incoherently, and frequently attempting to get out of bed, she became much quieter, but was extremely weak, her skin cold and clammy, and she spoke incoherently. Her excretions were all passed involuntarily. After lying in a comatose condition for some time, she expired on the morning of the 7th of August.

The body was examined by Mr. Prescott Hewitt.

The cranium contained a small quantity of fluid in the subarachnoid tissues; the brain was anæmic, but otherwise quite healthy. The kidneys were granular and small. The spinal cord seems not to have been examined.

ON A REMARKABLE CASE

OF

EXTREME MUSCULAR ATROPHY

WITH

EXTENSIVE DISEASE OF THE SPINAL CORD.

BY

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SAMUEL SOLLY, F.R.S., PRESIDENT.

NOTES OF THE CASE BY DR. JOHNSON.

Received June 9th.—Read June 23rd, 1868.

JAMES CORMACK, æt. 32, a peasant, was admitted into Kilkenny County Hospital, May, 1867. Obiit October, 1867. The appearances then presented were these. The muscles of the shoulder and arm had almost altogether disappeared. The muscles of the forearm remained apparently unaffected. The muscles of the ball of each thumb were almost altogether gone, while the muscles of the fingers continued to be well developed. The muscles on the dorsum of each scapula were nearly gone also.

The various organs of the pelvis and the lower extremities

were well developed. The virile organs were, as the man's aspect indicated, of full proportion, complete and perfect. The thighs and legs were muscular and full, *especially the thighs*, the muscles of which were particularly well developed. Whatever cause had affected the nutrition of other parts did not appear to have extended to them. His height was about 6 ft. 2 in.

The heart's action was extremely perturbed and irregular, and very feeble. It jumped and fluttered in a very remarkable manner, and any exertion, such as the ascent of stairs or incline, was attended with dyspnœa. The disturbance appeared to be functional, as there was no sound indicative of organic change or lesion of the heart. Notwithstanding the attenuated state of the arm, the brachial artery could not be found, while the radial was easily felt at the wrist.

The history of his case, so far as it could be ascertained, was, that he had been inoculated with the natural pock when about a year old. That he had been a very fine and healthy infant up to this time. That he suffered much after the inoculation and screamed at night, and gave other evidences of severe indisposition, and that, when the eruption was at about its height, the left arm was observed suddenly to droop and become powerless, and soon afterwards the right; and that both hung by his sides powerless for some time. It was a considerable time before he appeared to acquire any power in them. From this period the muscles already mentioned ceased to be developed, or fell into a state of progressive atrophy. *At the same time* the heart's action became irregular and perturbed, and continued so ever after.

Such was the account given by the patient himself. I had doubts as to his accuracy; but, *after his death*, his mother corroborated all his statements in a manner so clear, *positive*, and circumstantial, that I am inclined to think her representations not unfounded. She asserts most positively that he was free from all defects up to the time of the inoculation. That he was then a remarkably fine healthy child, with full use of his arms. That they suddenly became powerless and drooped helplessly by his sides, and that at the same time she at once

became conscious of the excited and irregular action of the heart, which she had never observed before.

On opening the thorax, the pericardium contained a considerable quantity of serum, but the heart was of about normal size, and the lungs crepitating, and apparently perfectly healthy. It is much to be regretted that the autopsy, which was made under circumstances of peculiar difficulty, was unavoidably hurried.

It seems remarkable that the same cause which arrested the nutrition of certain pairs of muscles did not affect the development of parts farther from the nervous centres; *e. g.* the lower extremities, as well as the pelvic organs, were wholly unaffected; and it may be asked, why did the muscles of the forearm and those of the fingers escape the blight which affected, in so remarkable a manner, the muscles of the thumb as well as those of the scapula and humerus? or why did not all parts *below* the seat of lesion suffer?

It would almost seem as if nature had expended the powers arrested in the upper extremities upon the development of the lower limbs. These were muscular and well nourished to more than an ordinary degree, while the *length* of the femur and tibia was out of proportion to the trunk. The thorax was small, and the chest narrow. The left side of thorax was larger than the right, causing a difference of height in the shoulders. The impression conveyed, *primâ facie*, was that the right side had fallen in, after the absorption of empyema, but he denied ever having had any illness which would indicate such a condition; and on opening the chest there was no trace, either on pleura or in lung, of past empyema, or disease whatever.

The abdomen was of ordinary proportion and development, and presented no indications of disease or arrest of due nutrition. The genital organs were fully proportioned. Had the development of the entire frame been equal to that of the lower part, I should say the man would have been 6 ft. 6 in. or 7 ft. high.

There was no curvation of spine. The difference in height of shoulders was caused by difference in size in the opposite

sides of the thorax. This, I think, must either have been congenital, or resulted from unequal nutrition, as he advanced to maturity; as I find no traces of lesion or disease to account for it.

It is obvious that the cause or causes which arrested the growth of certain muscles (for his mother asserts positively that they never became developed from the time the arms first fell powerless) did not arrest the development of other organs, certainly not of the bones, and especially not of the long bones.

The case seems to present many points of interest, and some difficult of solution.

Examination of the spinal cord, with remarks.

By Mr. Lockhart Clarke.

The greater part of the cervical and dorsal regions of the cord was placed in the museum of the College of Surgeons of Dublin, by Dr. Robert McDonnell, at the request of Dr. Johnson. In this part it appears that the *anterior* nerve-roots were in a state of extreme atrophy. Dr. Robert McDonnell has kindly furnished me with the following observations:—"The posterior nerve roots were normal; the anterior nerve-roots were in a state of extreme atrophy. The atrophy was quite marked and unmistakeable to the naked eye, but was made very striking on placing the portion of the cord examined side by side with a similar portion of a healthy cord. In the part examined microscopically by myself there were few nerve-tubules remaining in the roots."

I regret that only the lower third of the cervical enlargement was sent to me for examination. In this portion, however, I found the most unequivocal evidence of extensive lesions in both the white and grey substances. At its upper end the surface of the posterior columns was softened, and infiltrated with granular exudation, so that the posterior roots of the nerves were more or less destroyed, while the pia mater was thickened and also infiltrated with fluid exudation.

When this part of the cord was carefully hardened in

chromic acid, and transverse sections were examined, it was not difficult to perceive that extensive lesions and displacements of the grey substance had taken place. In Plate XI, fig. 1, the posterior horn on the left side has preserved its natural shape, with the gelatinous substance (*a*) forming a thin layer of paler grey substance at its extremity. On the right side, however, it was drawn backward into a long streak, between *a* and *á*, toward the softened surface of the corresponding posterior white column. On either side, the cervix cornu (*e*) or neck of the posterior horn was more or less damaged in different sections, by a streak of disintegration; and in the anterior grey substance there was an extensive area of softening and disintegration. On the left side, this was confined chiefly to the central parts (*f*), where the paler areas indicate the advanced and more transparent state of disintegration, while the dark isolated dots indicate the masses of tissue which are not yet reduced to that condition, but which, nevertheless, are damaged in a greater or less degree. On the right side the lesions in the *central* parts of the anterior grey substance are somewhat less conspicuous, but we find a dilated blood-vessel (*b*) with its branches, bordered by a thin layer of exudation, and by the surrounding tissue in a state of disintegration. The anterior median fissure (*d*), which always transmits a large blood-vessel, was dilated and filled with granular exudation, together with the *débris* of fibres at the inner borders of the anterior columns, resulting from disintegration.

A little higher up the grey substance was still further damaged and displaced, as represented in fig. 2. Here we find the same kind of exudation and *débris* of nerve-fibres filling up the anterior median fissure (*d*), and an area of the same nature surrounding a blood-vessel on each side of the anterior commissure. On the left side nearly the whole central portion of the anterior grey substance (*f*) was in a state of softening and disintegration, and a streak of the same kind extended backward along the middle of the posterior horn. On the right side the grey substance was very much altered in shape as well as damaged in structure. The

posterior horn was misshapen, the gelatinous substance being divided into two separate parts, *a á*. Between the anterior horn *b* and the head of the posterior horn *a*, the grey substance (*f*) was drawn out into the lateral column and extensively damaged by softening and disintegration of its tissue. The *tractus intermedio-lateralis, c*, was elongated in the same direction.[1]

Although some of the nerve-cells retained their normal size and appearance a great number were found to be in different states of atrophy and disintegration. In many instances the large cells of the anterior grey substance were reduced to at least the one-eighth or one-tenth of their ordinary size, and even to mere granular points; while in other instances, although not much reduced in size, they had lost their sharpness of outline and consisted of an uneven aggregation of granules. Dr. Robert McDonnell, who had also examined some parts of the cord, informs me that he "found abundant evidence of atrophy of the cells, although in some portions they were good."

In the ganglion which was sent me, belonging to the posterior nerve-roots, there was no apparent deviation from the normal state.

Remarks.—This is the eighth case of muscular atrophy in which I have examined the nervous centres supplying the affected muscles. In every case I have found unequivocal evidence of lesions of those centres, accompanied frequently by atrophy of particular parts of the grey substance, including the nerve-cells. Only one of these cases remains unpublished.[2] The various kinds of lesions and other abnormal appearances have been seen in my preparations by so many competent witnesses, both English and foreign, that no one now entertains the slightest doubt with regard to their

[1] The tractus intermedio-lateralis, which, when I discovered it in 1851, I so named from the fact of its being situated at the lateral boundary of the grey substance, and intermediate between the anterior and posterior horns, is a pale tract of grey substance connected with the spinal accessory nerve.

[2] The rest are as follow, with the dates and places of publication:

1. A remarkable case of muscular atrophy, with disintegration of the

existence. But there are some who think it not improbable, and there are others who are much inclined to believe, that these lesions and atrophies of the nervous centres and nerves are not primary affections, but consequences of the atrophy of the muscles which they supply. I cannot think that there is any good ground for this opinion. If the alterations in the nervous centres consisted only of *atrophy*, or diminution in size either of the grey or white substance, it might then, *à priori*, be justly considered as possible, or not improbable, that those alterations were the consequences of atrophy of the muscular tissue; and this supposition would be supported, to a certain extent, by the results of observation on cases in which limbs have been lost for a great number of years. Dr. Vulpian, of Paris, has recently published a very valuable article on this subject in its relation to the lesions and alterations which I have discovered in the nervous centres in cases of muscular atrophy.[1] He describes and represents the appearances that he found in the spinal cords of two persons in whom amputation had been performed at a very remote period. The first was a woman who at the age of

grey substance of the spinal cord. Plate. Beale's 'Archives of Medicine,' No. IX.

2. On a case of muscular atrophy, with disintegration of the grey substance of the spinal cord and atrophy of the cells. Engravings. 'British and Foreign Medico-Chirurgical Review,' July, 1862.

3. Examination of the spinal cord in a case of "wasting palsy." Displacement, atrophy, and disintegration of the grey substance of the cord. Ibid., October, 1863. Engravings.

4. A case of muscular atrophy, with extensive displacement and disintegration of the grey substance of the cord, and degeneration of nerve-cells. Five Plates. Beale's 'Archives of Medicine,' No. XIII.

5. A case of muscular atrophy, with disintegration of the grey substance of the cord, and enormous dilatation of the blood-vessels. Plate. 'Medico-Chirurgical Transactions,' 1866.

6. Another case, with disintegration of the grey substance and lateral white column, and remarkable atrophy of the nerve-cells. Ibid., 1867. Plate.

[1] Influence de l'abolition des fonctions des nerfs sur la région de la moëlle épinière qui leur donne origine.—Examen de la moëlle épinière dans des cas d'amputation d'ancienne date, par A. Vulpian, 'Archives de physiologie normale et pathologique,' No. III, Mai—Juin, 1868.

twelve years suffered amputation of the right leg, and who died of cerebral apoplexy at the age of fifty-nine, that is, forty-seven years after the operation. All parts of the cord were examined, but scarcely any appreciable difference was observed between the two lateral halves, either in the cervical or dorsal region, or even in the superior part of the dorso-lumbar enlargement. In these regions there was at most only a very slight diminution of the white substance of the right half; but in the *lower* part of the lumbar enlargement, nearly to the end, the right half of the cord was evidently smaller than the left. The grey substance and anterior column were the parts most atrophied; and the *posterior* column seemed to be the only portion of the right side that escaped; but the nerve-cells retained their normal appearance, nor was there any new formation of connective tissue either in the grey or white substance. Neither by the naked eye nor by means of the microscope could any appreciable difference be discovered between the nerve-roots of the two sides.

The second case was a woman who died of pleuro-pneumonia of the left side in 1868, and whose left leg was amputated, in 1848, a little above the ancle. The leg and thigh of that side were more slender than those of the right. The nerves were below their normal size, but no alteration of their fibres was observable under the microscope. On external examination there was no visible alteration in the cord, and both the posterior and anterior nerve-roots were similar on the two sides. On making sections of the cord, however, it was evident that the left side was smaller than the right in every region. This alteration was very slight at the lower extremity of the dorso-lumbar enlargement. A little higher up, but still below the middle of that enlargement, the diminution of the left side was much more conspicuous; while at its middle or thickest part the left side resumed its regular form. The anterior horn and white column, however, were a little smaller than those of the opposite side. In the dorsal region the only diminution of size was in the left anterior column; "but a more attentive examination," says

M. Vulpian, "shows that in the anterior grey substance on the left side there are three areas filled with a transparent substance, which in some places was a little granular,—areas which are evidently produced by an alteration of structure similar to that which Mr. Lockhart Clarke has discovered in different diseases, and amongst others, in progressive muscular atrophy." In the inferior part of the *cervical* enlargement there was diminution only of the left anterior column; but in the *middle* of that enlargement the whole of the white substance of that side had undergone a remarkable reduction in size, while the grey substance was but little changed.

The first opportunity I had of ascertaining whether amputation of a limb was followed, after many years, by atrophy of that portion of the cord from which it derives its nerves, was afforded me by my friend Dr. Dickinson, of St. George's Hospital, who very kindly gave me a portion of the lumbar enlargement of the cord of a seaman, aged seventy-four, whose left thigh had been amputated fifty-three years ago, in consequence of a cannon shot received in an action with the Danes in 1812. The man died in 1865. In the portion of the cord which I examined there was no difference in the size of the two lateral halves, as may be seen in fig. 3, Plate XI, which is an exact outline of the section taken by means of a camera lucida. The only difference was a very slight diminution of the left lateral grey substance (*g*); and the cells it contained were certainly less numerous and less distinct; but there was an entire absence of any alteration resembling the lesions and disintegrations which I have shown to exist in muscular atrophy. Dr. Dickinson, who also examined this portion as well as other portions of the cord, found no alteration in the lower lumbar region; but higher up in the lumbar and in the dorsal region there was marked diminution of the left posterior column, with an increase of connective tissue. The sciatic nerve, which he also examined, terminated at the stump in a large bulbous extremity, but the trunk of the nerve presented to the naked eye little or no alteration. From transverse sections of both sciatic nerves, which he prepared according to my method, and which he was kind enough to lend me, I

have made the drawings represented in figs. 4 and 5, Plate XI. Fig. 4 represents the ultimate tubules or fibres of the nerve in the sound leg, magnified 670 diameters; and fig. 6 represents those of the opposite stump. In the latter the originally large tubules were, for the most part, reduced to a number of small irregular areas surrounded by a close network of connective tissue, which contained a multitude of granules, resulting apparently from the débris or atrophy of the medullary sheaths or white substance.

Another case collected by Dr. Dickinson was that of a seaman who had his left arm amputated, two inches from the shoulder-joint, in the year 1842, and who died in 1865. From preparations which Dr. Dickinson kindly lent me I made the sketches represented in figures 6 and 7, Plate XI. In the cervical enlargement (fig. 6) no appreciable difference was observable in the dimensions either of the grey substance or the antero-lateral column (*h h'*) of the left side; nor was there any trace of lesion or disintegration; but the posterior column (*i i'*) was reduced to about two thirds the size of its fellow of the opposite side, and the fissure which, in this region, divides the column into two portions, contained an unusual quantity of connective tissue. Similar alterations were observed in the medulla oblongata, on a level with the lower part of the decussation of the anterior pyramids, as shown at *i i'*, fig. 7. Above this point the posterior column resumed its normal dimensions.[1]

Since, therefore, both the portion of the cord and the nerves which supplied a lost limb will undergo, after a number of years, a very decided atrophy—although, apparently, in different places, to a very different extent—it is not improbable that *atrophy* of muscles, progressing through a lengthened period, might give rise, through partial abolition of the functions of the nerves and nervous centres which supply them, to *simple atrophy* of these parts. But it is highly improbable that muscular atrophy is the cause of

[1] In the forthcoming number (November, 1868) of the 'Journal of Anatomy and Physiology,' a full account of Dr. Dickinson's investigations will be published.

those lesions of structure, of those morbid exudations, those remarkable areas of transparent fluid or granular disintegration, or of that enormous dilatation of blood-vessels, which I have discovered in the cords of so many cases of this disease; and the absence of such lesions in cases of lost limbs is strongly in favour of this conclusion. It is true, as I have already shown, that M. Vulpian detected in one solitary spot what appeared to him to be three small areas of disintegration. This, however, not only seems to be an exceptional instance, but was found far distant from that portion of the cord which supplied nerves to the lost limb. It is evident that M. Vulpian laid no stress upon this fact, for he concludes his examinations in the following words: "De plus, et c'est ce que M. Lockhart Clarke a déjà mis en évidence, il n'y a aucune analogie entre les lésions qu'il a décrites dans l'atrophie musculaire progressive et cette simple diminution de volume des diverses parties de la moelle constatée dans des cas d'amputation d'ancienne date. *Nous pouvons donc conclure que les lésions medullaires de l'atrophie musculaire reconnaissent une autre cause que la seule paralysie des muscles et des nerfs.*"

There is abundant evidence to prove that a morbid condition of nerves depending on pressure, wounds, or some other kind of irritation, is the cause of various diseases of the skin, and even ulceration of the subjacent tissues. The writings of Henle,[1] Rayer,[2] Barensprung,[3] Notta,[4] Swan,[5] Parrot,[6] Charcot,[7] Paget,[8] Earle,[9] and others,

[1] Henle, 'Handbuch Ration. Pathologie.'

[2] Rayer, 'Maladies de la Peau.'

[3] Baerensprung, 'Annalen der Charité Krankenhauser zür Berlin.'

[4] Notta, "Mémoire sur les lésions fonctionnelles qui sont sous la dépendance des névralgies," 'Archives Generales de Méd.,' 1854.

[5] Swan, 'Diseases and Injuries of the Nerves.'

[6] Parrot, "Consideration sur le zona," 'Union Médicale,' Mars, 1856.

[7] Charcot, "Note sur quelques cas d'affection de la Peau dépendant d'une influence du système nerveux," 'Journal de Physiologie,' tome deuxième, 1859.

[8] Paget, 'Lectures on Surgical Pathology.'

[9] 'Medico-Chirurgical Transactions,' vol. vii.

record numerous instances of this kind. It is well known how commonly neuralgia is followed and accompanied by *herpes*, which sometimes takes the course of particular nerves, as the intercostal or lumbar, and Dr. Anstie has shown that it is sometimes followed by erysipelas. Dr. Hugh Ley long since remarked that leech bites induce erythema and ulcers in the skin of neuralgic subjects. Rayer records a very interesting case in which traumatic neuralgia, arising from a gunshot-wound in the thigh, was accompanied at intervals by herpetic eruptions on the skin. Rouget has related a similar case.[1] In like manner the process of nutrition in muscles may be perverted and impaired by disease or injury of their nerves. Every practitioner of experience must have met with cases in which groups of muscles have wasted, sometimes to a remarkable degree, from this cause alone. Two striking instances recently came under my own observation. One was a boy, who slipped from the edge of a high stool on which he was rocking himself, and fell heavily to the ground on one side of the sacrum and of the lower part of the spinal column. For some days he complained very little; but, soon after, the glutei muscles, as well as those of the loins on that side, rapidly wasted to an extraordinary degree, so as to cause lateral curvature of the spinal column by the superior force of the corresponding muscles of the opposite side. The other case was a gentleman who was knocked down by a cab, one of the wheels passing over the *front* of his shoulder. The swelling for some time was very great, but when this subsided the muscles of the shoulder rapidly wasted. The deltoid of this patient, who was a powerful and muscular man, almost wholly disappeared, so that the outer part of the shoulder was perfectly flat, and even somewhat sunk below the acromion process, as in dislocation of the head of the humerus into the axilla. Both these cases rapidly recovered by the application of blisters to the affected and tender parts.

Now, if functional derangement or irritation of nerves will

[1] See also a paper by Dr. Woakes, in the 'Journal of Cutaneous Medicine,' 1867.

give rise to atrophy of the muscles which they supply, it is reasonable to infer that some kind of functional derangement of the nervous centres, which give origin to those nerves, may be followed by similar effects; so that although there are the strongest grounds for believing that the lesions of *structure* which I have described in the nervous centres of cases of muscular atrophy are the cause of that atrophy, it does not therefore follow that every case of muscular atrophy is the consequence of those *structural* lesions, or even of *functional* disturbance of the nervous centres, since it may be due to affections of the nerves alone.

In the history of the singular case which forms the subject of this communication we learn that after inoculation, at the age of twelve months, the child screamed at night and gave evidence of severe indisposition; that when the eruption was at its height the left arm was observed suddenly to droop and become powerless; that, soon after, the right arm followed the same course; and that it was long before the patient seemed to acquire any power in them. At the same time the heart's action became irregular and continued so ever after. These symptoms were followed by atrophy of the muscles of the arms and shoulders. If these statements be true, they point in a very remarkable manner to disease of the spinal cord as the cause of the muscular atrophy; for it is evident, 1st, that the pustules of inoculation on the cutaneous surface of the arms could have had no share in paralysing the muscles, except by inducing some morbid change in that portion of the cord from which their motor nerves were derived; 2nd, that they could have had no share in causing *atrophy* of those muscles, and of the muscles about the chest and back, except through the same channel.

M. Vulpian performed some experiments to ascertain whether any effects were produced on the nutrition of muscles by injuries inflicted on the grey substance of the spinal cord. He found that these injuries were not followed by atrophy of the muscles, which receive their nerves from the injured part; and he therefore concludes that if there be a close relation of causality between the lesions of the

cord and those of the muscles, in muscular atrophy, we may presume that the influence of the spinal lesions depends on their peculiar irritating nature. The observations of Brown-Séquard confirm this opinion. In commenting on M. Charcot's cases of affections of the skin depending on the influence of the nervous system, he shows the difference of effects between *simple section*, or *cessation of action*, of nerves, and their *irritation*. Simple section of a nerve is not followed by any other alteration of nutrition than slow atrophy of the paralysed parts. It is the same with the spinal cord. He has seen animals live for months after section of the spinal cord without presenting any other lesion of nutrition than a slow atrophy of the paralysed parts; whereas, in two cases in which exostoses formed, at the point where the cord was divided, and compressed its lower end, considerable atrophy of the muscles supervened in the course of five or six *days*, with gangrenous ulceration over the sacrum and some parts of the thigh.[1]

In making these remarks on the cause of muscular atrophy, I have been influenced solely by the great preponderance of evidence in favour of one view rather than the other. I have not been contending for a favorite theory; and as my object is solely to arrive at the truth, I would strongly urge a searching investigation into every case that may seem capable of throwing light on this important subject, until every question of doubt be definitively settled.

[1] 'Journal de Physiologie,' tome deuxième, 1859, p. 112.

EXPLANATION OF PLATE XI.

FIG. 1. Transverse section of the grey substance of the cord in the cervical enlargement; *a a'*, the gelatinous substance of the posterior cornu; *b b*, the anterior cornua; *c*, the tractus intermedio-lateralis; *d*, granular exudation filling up the anterior median fissure; *e*, the cervix cornu or neck of the posterior horn; *b'*, a dilated blood-vessel in the middle of the anterior grey substance; disintegration of the central part of the grey substance of left side; the lighter parts on the opposite side indicate the same kind of alterations.

Fig. 2. The same letters indicate the same parts; on the right side the lateral grey substance (*f*), and the tractus intermedio-lateralis (*c*) are drawn out into the lateral column.

Fig. 3. Transverse section of the lumbar enlargement of the spinal cord of a seaman who suffered amputation of the left leg fifty-three years before his death. . Only a trifling degree of atrophy of the left anterior grey substance was discovered at *g*.

Fig. 4. Part of a transverse section of the great sciatic nerve of the sound limb, from the same man, showing the size and general appearance of the cut-ends of the nerve-tubes with the axis cylinders in their centres, and united by connective tissue; magnified 670 diameters.

Fig. 5. Part of a similar section from the great sciatic nerve in the stump of the amputated leg, showing how the originally large nerve-tubes have been reduced to a number of small irregular spaces, with intervening granules; magnified 670 diameters.

Fig. 6. Transverse section of the cervical enlargement of the spinal cord, from a seaman who had suffered amputation of his left arm two inches from the shoulder twenty-three years before his death. The only part atrophied was the posterior column on the left side, *i i'*.

Fig. 7. Transverse section of the lower part of the medulla oblongata, a little above the points of the anterior pyramids from the same man; *b b*, anterior grey cornu; *k, k*, caput cornu posterioris or grey tubercle of Rolando; *l*, central grey substance; *m, m*, anterior pyramids, with their decussating fibres between the anterior cornua, *b b*; as in preceding figure, the left posterior column *i i'* is the only part atrophied.

Fig. 1.

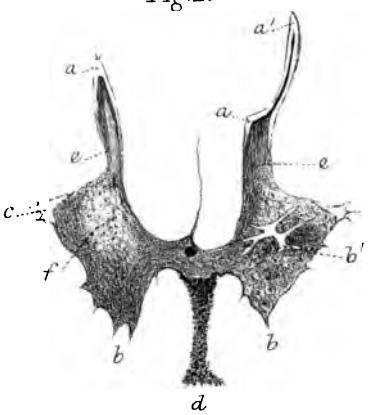


Fig. 2.

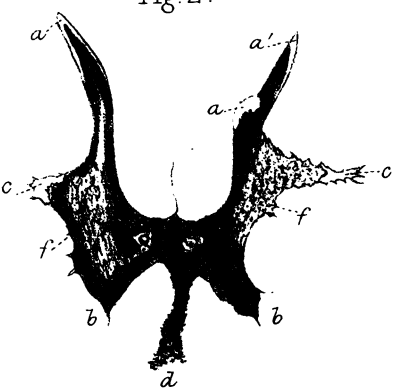
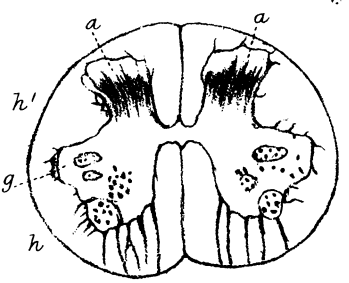


Fig. 5.

Fig. 3.



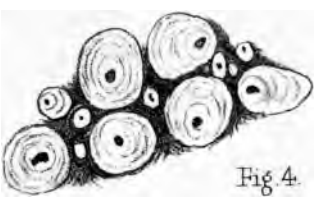
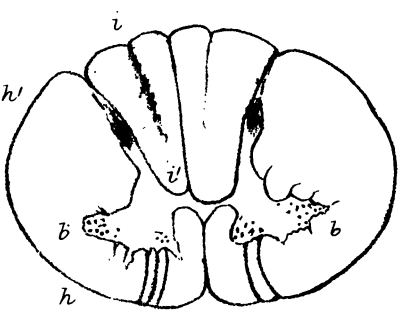


Fig. 4.

Fig. 6.



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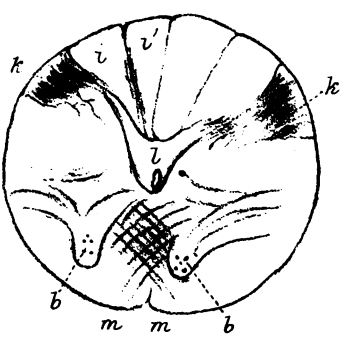

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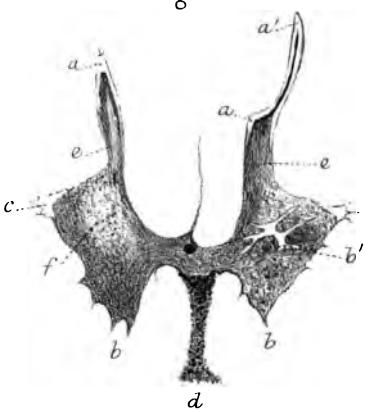


Fig. 2.

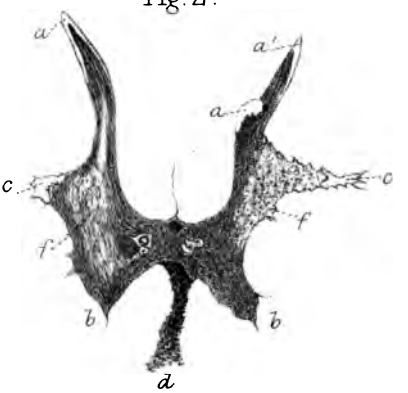


Fig. 5.

Fig. 3.

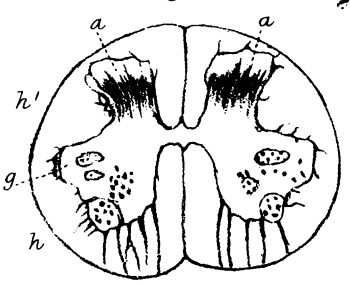


Fig. 4.

Fig. 6.

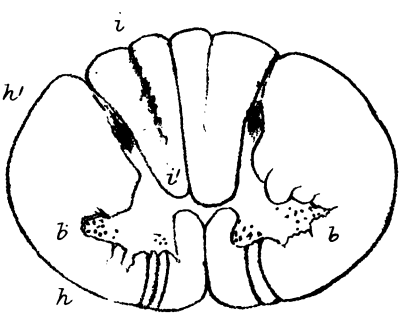
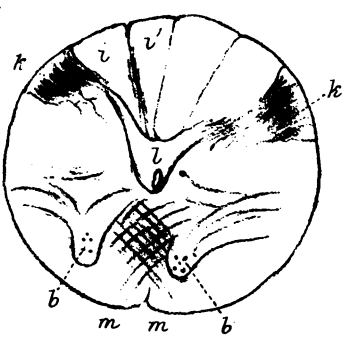


Fig. 7.



DESCRIPTION

OF THE

SPINAL CORD IN A CASE OF TRAUMATIC TETANUS.

BY

W. H. DICKINSON, M.D. CANTAB., F.R.C.P.,

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Received June 9th.—Read June 23rd, 1868.

IN a case of traumatic tetanus which recently proved fatal in St. George's Hospital, the spinal cord presented some striking pathological changes which may interest the Society, more especially as Mr. Lockhart Clarke has in a recent paper ('Medico-Chirurgical Transactions,' vol. 48) drawn attention to alterations of the same nature in several similar instances.

A carman, 25 years of age, was admitted, under the care of Mr. Holmes, on the 14th of last January, with a wound which had been recently inflicted by an iron spike through the palm of the left hand. Both orifices of the wound presented a jagged outline. The metacarpal bones had escaped injury. The wound was dressed with carbolic acid paste, and the patient, who was able to walk about and eat well, went on favorably until the morning of the 22nd. At 4 a.m. on that day he was disturbed by a feeling of pain and stiffness about

the jaws. By 11 he was in a state of decided opisthotonos. The head was thrown back, he was scarcely able to open his mouth; the muscles of the neck, spinal region, and abdomen, were rigid; and he complained of pain down the back. Tetanic spasms now occurred with great frequency, having scarcely any interval between them; and at 10.30 in the evening he died, eighteen and a half hours after the appearance of the first tetanic symptoms. The treatment consisted of the subcutaneous injection of morphia and atropia.

The foregoing history was obtained from notes taken by Mr. Leigh, the surgical registrar.

The post-mortem examination was made by Mr. Pick, who supplied the following details excepting those which relate to the cord and nerves, which are from my own observation.

The examination took place thirty-five hours after death; the weather was cold, and there were no signs of putrefaction about the body.

The wound was in a sloughing condition. It had severed one of the branches of the median nerve. The divided nerve had a pink tinge near the injury, but when subsequently the nerve was examined in section by the same process as was used for the cord no departure from the natural state was detected.

The lungs were congested and friable. The heart was empty, both ventricles being closely contracted. The valves and muscular structure were natural. The liver was congested, but not otherwise altered. The spleen was natural. The kidneys were somewhat rough on their surfaces, their capsules were adherent, and their cortical portions slightly shrunk.

The spinal dura mater was remarkably injected, as also was the pia mater, the cord being covered along its whole length with a network of conspicuous red vessels.

The cord was, in three places, altered in a striking manner in its external configuration. On the right side of the cervical enlargement was an oval swelling about the size and shape of a split bean, which lay between the roots of the nerves, pushing the anterior roots a little forward, the posterior a little back. This protuberance measured about a third of an

inch in the vertical direction, rather less from before backwards, while the eighth of an inch was about the extent of its lateral projection. In colour and external appearance the swelling did not differ from the neighbouring surface.

Opposite the first lumbar vertebra a more extensive change of the same kind was found. The cord was here expanded on its posterior aspect into a flat swelling which nearly doubled its natural width and was obvious by the distension it occasioned, before the dura mater was laid open. The swelling was about an inch in length. It was confined to the posterior aspect of the cord, of which both sides were involved, the left more than the right. The surface of the swelling was somewhat lobulated. In colour it resembled the adjoining part of the cord, excepting that the injected vessels upon it were more closely placed, and that there were many specks of extravasated blood showing through the pia mater.

About half way between this tumefaction and the lower end of the cord was a smaller circumscribed prominence of the same nature, which lay likewise on the posterior aspect of the cord. It was hemispherical, of about the size of half a large pea, and lay close to one of the left posterior roots, between it and the median fissure.

The cord was now subjected to microscopic examination in order to ascertain the minute changes which had led to these remarkable alterations of shape. Various portions were examined in their fresh state and in glycerine, while sections were made at intervals by the method introduced by Mr. Lockhart Clarke.

This method of preparation, better adapted than any other to show minute changes of structure while maintaining the continuity of the tissues, involves soaking in a solution of chromic acid, which not only hardens the nervous matter, but solidifies the contents of the blood-vessels as well as any serous or albuminous effusions which may occur. It was, therefore, easy to note any excess of blood in the tissue, and also to detect fluid exudations which under other circumstances might have escaped notice.

The changes about to be detailed are, firstly, a remarkable

injection of the blood-vessels in certain parts of the cord, by which they became replete to distension with their natural contents, while in a few situations blood-corpuscles had escaped from their proper channels and diffused themselves among the nervous elements. Secondly, a structureless transparent material had been poured out in the immediate vicinity of the vessels, not only into such vacant spaces as exist in the fissures of the cord, but forcibly intruded into many parts of the solid structure, tearing up the tissue and displacing the neighbouring parts. Beside these changes, which affected both the white and grey matter, the white columns presented circumscribed alterations, which, in conjunction with the effusion described, caused the swellings which were so conspicuous on the surface of the cord. The changes indicated may now be described more particularly, taking the cord in position from above downwards. It may be stated that the medulla oblongata, which to the naked eye appeared natural, was not preserved for examination.

In the uppermost part of the cervical region many of the blood-vessels were distended to two or three times their proper width, and formed conspicuous objects. This was the case in the anterior fissure and throughout the whole grey matter. The left posterior horn was more injected than any other part, and in this situation there were several spots where blood-corpuscles had escaped from a vessel and spread themselves in its vicinity. Between the more injected posterior horn and the central fissure was a point where the pia mater had given way, forming a small opening through which the nerve-fibres streamed out in a centrifugal direction, forming a small hernial protrusion evident enough under the microscope, though barely visible to the naked eye.

On reaching the cervical enlargement, which was the seat of the external swelling first mentioned, the same distension of the blood-vessels continued, with the addition of a remarkable mass of smooth homogeneous matter in the central part of the right grey crescent. This new formation lay in a torn cavity in the midst of the grey matter, and had displaced its lateral portion outwards in such a manner as to

cause the superficial projection which has been described. The intruded material measured about one eighth of an inch from before backwards, and reached more than twice as far vertically. It consisted of a perfectly structureless and translucent substance, which took a brilliant pink tint, with carmine, distinguishing it from the nerve-matter around it. It was irregular in shape, filling a rent which had several cracks reaching from its corners towards the front, back, and lateral boundary of the grey matter. The lateral portion of the grey matter was pushed outwards, and the central fissure was bent into a curve, with its concavity towards the left side.

At the outer edge of the rent was a small extravasation of blood, which had imparted a rusty stain to the adjoining part of the cord.

Beside the large mass of new material in the right horn, were two or three much smaller patches scattered mostly in the left grey matter in or near the commissure. The exudations generally lay on the outside of a blood-vessel, the tissues of the cord being torn to a greater or less extent, as if fluid had been forcibly impelled into it.

The patches of new material generally imbedded, particularly at their edges, a few globules, threads, or finely granular matter, the débris of nerve-structure, while the adjacent tissue often had a frayed look, nerve-fibres hanging with loose ends, as if they had been partially dissolved or corroded by the effusion.

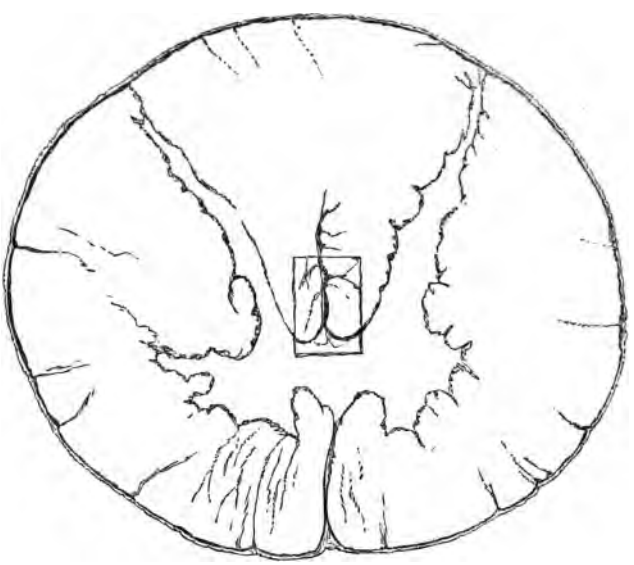
(Plate XII, fig. 1, represents a section through the middle of the swelling described, and shows the exudation in the right side, and consequent distortion of the cord.)

In the lower part of the cervical enlargement the cord again became symmetrical, owing to the absence of the larger mass of adventitious material, though many smaller patches were scattered through the grey matter and fissures.

The state of blood-vessels which has been described in the upper part of the cervical region continued here, and persisted throughout the whole of the cord, the structure

being traversed by swollen and replete vessels, the grey matter being more injected than the white, and the left posterior horn more than any other part. Vessels distended in the same manner also existed generally, in both anterior and posterior fissures.

In the lower part of the cervical region the vessels in the anterior and posterior fissures were in places surrounded by the exudation, which was in some situations sufficiently abundant to fill the entire space between the vessel and the walls of the fissure, and even to encroach here and there upon the nervous matter, which had been disintegrated and frayed out at the point of contact. (See Plate XII, fig. 2.)



Exudation in fissure of the cord in connection with a blood-vessel.

This is given to show the exact position of the portion which is shown enlarged on Plate XII, fig. 2.

Quite at the lower part of the cervical enlargement was a hernial protrusion, in contact with the left posterior horn, a

mass of nerve-tubes streaming out centrifugally through a rent in the pia mater, forming a small abrupt swelling of about an eighth of an inch prominence, immediately in front of the emergence of the nerve.

From the lower end of the cervical enlargement to the beginning of the lumbar the changes were less conspicuous. The blood-vessels were in many places surrounded by such material as has been described, more or less intermixed with broken nerve-tubes. The central part of the dorsal region had undergone most alteration, being irregularly traversed by small fissures, which contained the intruded material.

In the lumbar enlargement the cord presented alterations of such extent as to cause the large external swelling which has been described. On making sections through the swollen part, it was found that both the grey matter and the white had contributed to produce the change of shape.

In the right crescent was a large mass of exudation, such as has been described in the cervical region, while in the left was a smaller blotch of extravasated blood. Each of these intruded materials lay in a torn cavity in the middle and posterior part of the grey matter.

The exudation in the right horn was uniform, translucent, and abruptly circumscribed, like that in the cervical region, which it closely resembled. On the left side the tearing of the cord was extensive and irregular, a rent nearly an eighth of an inch long traversing the grey matter from before backwards. This laceration contained extravasated blood, and blood-corpuscles were also diffused into the surrounding tissue, and blended with the white matter. A large distended vessel, in which, however, no rupture could be detected, lay in the torn cavity described. Beside the extravasated blood, a minute quantity of translucent exudation was here seen.

By the exudation on one side and the extravasation on the other the hinder extremities of the grey matter were displaced, so as to contribute largely to the external swelling. There was, however, a coincident change in the white matter, in the posterior and lateral columns. These, more particularly the left lateral column, were increased in bulk by a

swelling in the nerve-fibres composing them, the outer portions of which were thicker than was the case elsewhere. Beside this, the fibres were separated from each other by an opaque granular material. In one place also, close to the tip of the left posterior horn, was a considerable mass of finely granular substance, which existed to the exclusion of nerve-tubes, and took a pink colour with carmine. One or two smaller patches of the same nature were found in other parts of the posterior columns.

The changes described became less extensive towards the upper and lower limits of the tumefaction, where the cord resumed its natural size; nothing further was noticed than the distension of the vessels which has been so often referred to. The same statement applies to the rest of the cord, with the exception of the small space where the hemispherical protuberance which has been described lay upon the surface of the left posterior column.

This occurred close to the emergence of one of the posterior roots. The drawing (Plate XIII, figs. 1 and 2) represents the state of the cord opposite to the middle of the swelling. The cord is here distorted, the central fissure bent, the grey matter rendered unsymmetrical by an elongation of the left posterior horn, while the corresponding posterior column is swollen so as to elevate the surface and cause the prominence described. The immediate cause of the swelling appeared to be a large exudation of the same translucent, structureless, carmine-absorbing material which has been described in other parts of the cord. This effusion, distinguished in the drawing by its pink colour, lay as a large irregular mass in the centre of the tumefaction, where it existed for a considerable space unmixed with any organized structure. Beside this large pool of exudation, which was visible to the naked eye, were several small patches of the same substance, some of which imbedded globules of disintegrated nerve-fibre; and a similar exudation was also intimately diffused among the nerve-fibres, between which it had insinuated itself. A narrow stream of the same material filled the posterior fissures and imbedded broken-down remains of the nervous

elements. A similar effusion was intimately spread among the fibres of the posterior horn, and a considerable mass of the same nature lay on the outside of the cord, close to the attachment of the posterior root.

Where the nerve-tubes came in contact with the exudation they were mostly changed, as in other parts of the cord, as if by partial solution. In some parts of the swelling they had lost the distinction, ordinarily so clear, between the outer white matter and the axis-cylinder, while there were places where the tubular character of the tissue had almost disappeared, being replaced by a nearly uniform surface, showing but the faintest trace of the original structure.

Below the lowest tumefaction no morbid changes were detected, save the distension of the blood-vessels.

Without presuming that we have found the first disturbance of the nervous system which leads to the development of tetanus, we have recognised very definite lesions which are associated with it. The changes observed in this instance were probably of very unusual extent, since the swellings upon the cord were too conspicuous to have remained unseen, were they frequent in such cases.

The microscopic appearances are very suggestive. The blood-vessels appear to be, if not the first, at least an early seat of change. Distended with blood, not only to the uttermost of their natural capacity, but dilated to many times their proper width, and crammed with blood-corpuscles so as to look like solid cylinders, their condition gave evidence of an altogether abnormal relation between the pressure of the blood and the resistance of the walls. Either blood had been propelled into them with supernatural force, or, what is more likely, the tension of their coats had been lessened by a change in their innervation. The overcharge of the vessels led to the escape of their contents. In some places blood-corpuscles were extruded. More often only the fluid portions of the blood traversed the wall, to appear as the translucent structureless material which played so prominent a part in the destruction of the cord. That this translucent material was an

exudation from the vessels, and not a product of disintegration of tissue, is evident from the following facts. It constantly lay in contact with the vessels, and often surrounded them. It held the most changeable relations to the nervous matter, lying in the grey matter, in the white, abundantly in the fissures, and occasionally outside the cord altogether. It was the source of increase of bulk, of laceration, and of displacement, such as to suggest that it was an addition to the structure, and had been forcibly driven into it.

At the same time a certain amount of disintegration of the nervous elements had taken place, where the exudation came into contact with them, such as might result if, as suggested by Mr. Lockhart Clarke, the exudation had a solvent action upon the tissue.

It is probable that the tendency to disintegration in the nervous matter may have been enhanced by the unnatural state of the blood-vessels, and the consequent imperfect nutrition of the cord.

There are certain points of interest connected with the situation of the various lesions. The irritation from the left hand, conveyed, as we must suppose, by certain of the left posterior roots, occasioned especial congestion of the left posterior horn, and further changes in the white matter in contact with it—that is, in the left posterior and lateral columns. The central and anterior parts of the grey matter were most extensively affected on the side opposite to that of the injury, as might have been anticipated from the decussation in the cord of the sensory fibres. The irritation, having reached any column or segment of the cord, appeared to diffuse itself throughout its whole length with undiminished intensity. Although the cervical region must have been the first recipient of the morbid influence, the lumbar part of the cord, both in the white and grey matter, was at least as severely affected.

Postscript.—It may be well to state that the distension and repletion of the blood-vessels throughout the cord involved both arteries and veins, and, in a less degree, the capillaries.

The arteries, in their swollen state, varied up to $\frac{1}{230}$th of an inch in diameter, the veins up to $\frac{1}{150}$th. Both arteries and veins in this state were found in the fissures, as well as in the substance of the cord. Capillaries similarly packed with blood-corpuscles, measuring about $\frac{1}{3000}$th of an inch in diameter, occurred here and there in the grey and white matter. The measurements, given since they were obtained from portions of cord which had been subjected to the action of chromic acid and turpentine, are probably short of the real dimensions of the vessels. The process used commonly produces more or less contraction in the tissues subjected to it.

The exudation which has been spoken of was in most frequent connection with the arteries; there was found, indeed, only one clear instance of that formation in connection with a vein.

DESCRIPTION OF PLATES XII AND XIII.

PLATE XII.

Fig. 1 represents exudation in the grey matter of the cord in a case of tetanus (cervical region), × 7.

Fig. 2. Exudation in fissure of cord, in connection with a blood-vessel. (The pink tint shows the effect of carmine on the exudation-matter.)

PLATE XIII.

Fig. 1. Section through the swelling described in lumbar region, × 7.

Fig. 2. A small portion of fig. 1 more magnified, × 50.

The pink tint shows the effect of carmine on the exudation-matter.

Fig 1.

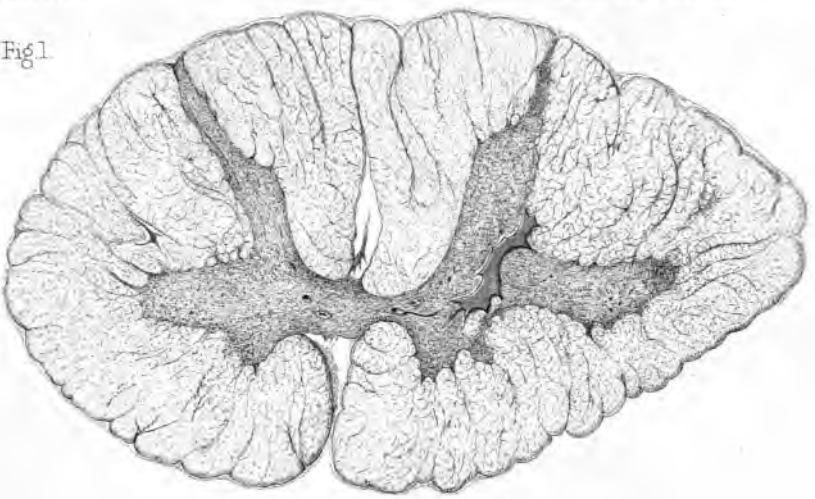


Fig 2.

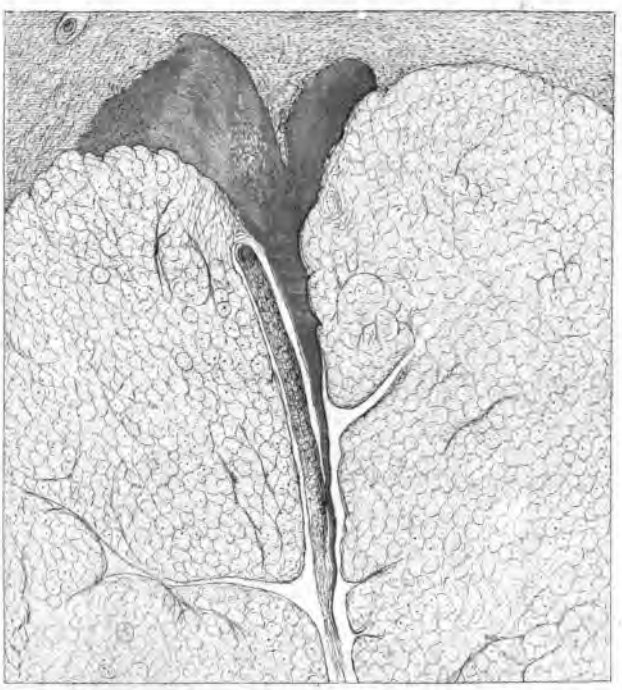


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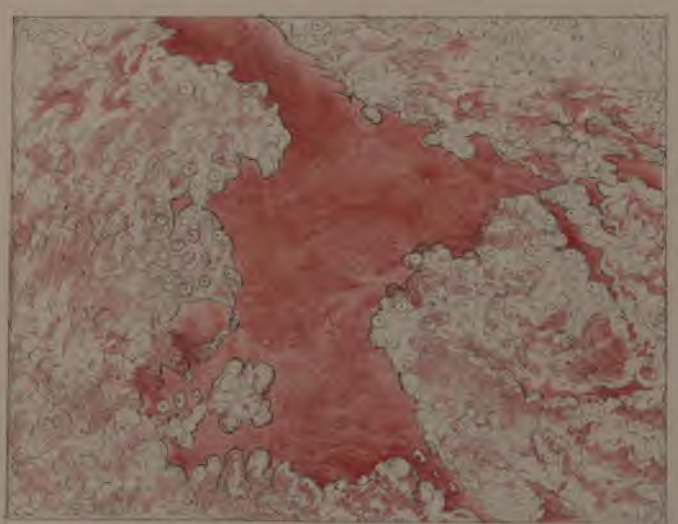
Fig 1.

Fig 2



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A STUDY

OF THE

INFLUENCE OF WEATHER AND SEASON UPON PUBLIC HEALTH, &c.

II. A COMPARISON OF THE INFLUENCE OF VARIATION OF TEMPERATURE UPON GENERAL SICKNESS IN THE COLDER AND WARMER SEASONS OF THE YEAR.

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In my former communication I pointed out the influence exerted by rises of mean temperature in increasing general sickness, and by falls in decreasing it, and endeavoured to establish the limits within which this operation is exercised. The question now arises, whether this influence is exerted in a similar manner and to an equal extent in the colder and warmer seasons of the year.

I may say here at the outset (for the fact is apparent on the most superficial view of the tables) that, whether we regard the frequency with which sickness occurred with rises of mean temperature and decreases with falls, or whether we regard the clear gain by way of increase as the result of the conditions operating in the weeks of rise or by way of decrease in the weeks of fall, or the amount of increase or

decrease of sickness in each set of weeks, the grand result is that, on the whole, the association of increase of sickness with rises, and of decrease of sickness with falls, is very much more marked in the colder than in the warmer seasons of the year.

I do not think I shall be far wrong in attributing this remarkable result to an essential difference in the signification of the term "mean temperature," when applied to these weeks respectively, and in considering that it is closely connected with the question of the diurnal range of atmospheric temperature. In the colder season of the year the diurnal range is comparatively small; in the warmer it is comparatively great. Hence in the colder season the "mean temperature" represents more closely the real temperature during all the twenty-four hours of each day of which a week consists; in the warmer season it represents it less closely, and, whatever the day temperature may be, its operation would be greatly modified by the lessened temperature of the night.

We may first of all compare the proportion borne by the cases of increase of sickness to those of decrease in the weeks of rise, and the proportion borne by the cases of decrease of sickness to those of increase in the weeks of fall, as exhibited in the Tables I and II, for the colder and warmer seasons respectively. I draw my line at 50°, the average temperature of the colder weeks of rise being 43·2°, and of the warmer 58·9°, and the average temperature of the colder weeks of fall being 40·6°, and of the warmer 57·1°.

1st.—As respects the weeks of rise of mean temperature.

For every 1000 cases of increase of sickness there occurred—

In the course of the 88 *colder* weeks of rise generally			345	cases of decrease of sickness.
„	108 *warmer*	„	620	„
„	28 *colder*	slight rise	1083	„
„	42 *warmer*	„	716	„
„	33 *colder*	moderate rise	285	„
„	40 *warmer*	„	1022	„
„	27 *colder*	considerable rise	123	„
„	26 *warmer*	„	177	„

Tables showing ness at mean temperatures below and above 50°.

Rises.	No. of weeks.	Mean temperature.	Sickness.			Neither Increase nor Decrease of Sickness.				
			Frequency of decrease per cent. of weeks.	Amount of decreased sickness. — Cases.		No. of weeks.	Mean temperature.	Sum of rises.	Mean weekly rise.	Frequency per cent. of weeks.
				Sum.	Mean.					
Generally ...	88	4	32·95	797	27·4	—	—	—	—	—
Slight.....	28	4	42·85	439	36·5	—	—	—	—	—
Moderate ...	33	4	36·36	218	18·1	—	—	—	—	—
Considerable.	27	4	18·51	140	28·0	—	—	—	—	—
Generally ...	108	5	41·66	1203	26·7	1	63·5°	3·5°	3·5°	0·92
Slight.....	42	5	42·85	545	30·2	—	—	—	—	—
Moderate ...	40	5	50·00	543	27·1	1	63·5°	3·5°	3·5°	2·50
Considerable.	26	5	26·92	115	16·4	—	—	—	—	—

Falls.	No. of weeks.	Mean temperature.	Sickness.			Neither Increase nor Decrease of Sickness.				
			Frequency of decrease per cent. of weeks.	Amount of decreased sickness. — Cases.		No. of weeks.	Mean temperature.	Sum of falls.	Mean weekly fall.	Frequency per cent. of weeks.
				Sum.	Mean.					
Generally ...	103	4	68·93	3074	43·2	1	44·1°	0·7°	0·70°	0·97
Slight.....	22	4	63·63	582	41·5	1	44·1°	0·7°	0·70°	4·54
Moderate ...	49	4	71·42	1289	36·8	—	—	—	—	—
Considerable.	32	4	68·75	1203	54·6	—	—	—	—	—
Generally ...	90	5	46·66	1464	34·8	3	55·8°	2·4°	0·80°	3·33
Slight.....	36	5	41·66	378	25·2	2	55·8°	0·4°	0·2°	5·55
Moderate ...	38	5	44·73	534	31·4	1	56·0°	2·0°	2·0°	2·63
Considerable.	16	5	62·50	552	55·2	—	—	—	—	—

2nd.—As respects the weeks of fall of mean temperature. For every 1000 cases of decrease of sickness there occurred—

In the course of the 103	*colder*	weeks of fall	generally 300	cases of increase of sickness.
„	90 *warmer*	„	907	„
„	22 *colder*	slight fall	572	„
„	36 *warmer*	„	1833	„
„	49 *colder*	moderate fall	315	„
„	38 *warmer*	„	838	„
„	32 *colder*	considerable fall	152	„
„	16 *warmer*	„	338	„

From which it is apparent that in weeks of rise of mean temperature (with the solitary exception of those of slight rise) the tendency to increase of sickness was greater in the colder than in the warmer weeks; and that in the weeks of fall of mean temperature, whatever the extent of the fall, the tendency to decrease of sickness was greater in the colder than the warmer weeks.

RISES OF MEAN TEMPERATURE.

In the colder weeks of the year a rise of mean temperature was associated with increase of sickness more frequently than in the warmer weeks; that is, in 67·04 per cent. of the colder, and in 57·4 per cent. of the warmer weeks. In the following Table III, which relates to the clear gain by way of increase of sickness, I estimate, as in my former paper, 1 degree of rise or fall of mean temperature as equivalent, normally, to 3·16 cases of increase or decrease of sickness.

TABLE III.—*General results of the* 88 *rises in weeks with a mean temperature below* 50°.

Rises.	Number of rises.	Total extent in degrees.	Estimated clear gain by way of increase of sickness.	Actual clear gain by way of increase of sickness.	Number of cases of increase above or below estimate.	Number of degrees of rise of temperature represented by the difference of estimated and actual increase.	
			Cases.	Cases.	Cases.	Sum.	Weekly mean.
Generally ...	88	366·0°	1156	1509	+ 353	+111·70°	+ 1·26°
Slight.....	28	28·4°	89	...	— 123	— 38·92°	— 1·39°
Moderate ...	33	117·2°	370	546	+ 176	+ 55·69°	+ 1·71°
Considerable.	27	220·4°	696	997	+ 301	+ 95·25°	+ 3·52°

General results of the 108 *rises in weeks with a mean temperature of* 50° *and upwards.*

Generally ...	108	367·5°	1161	737	— 424	—134·17°	— 1·24°
Slight.....	42	42·3°	133	216	+ 73	+ 23·10°	+ 0·55°
Moderate ...	40	127·8°	403	...	— 415	—131·32°	— 3·28°
Considerable.	26	197·4°	623	533	— 90	— 28·48°	— 1·09°

The clear gain by way of increase of sickness in the colder weeks of rise of mean temperature was more than twice as large as in the warmer weeks, although the total sum of rises in each series was nearly the same. In the 88 *colder* weeks the clear gain exceeded considerably that which, according to my tables, might have been anticipated, the excess being such as might have been looked for had the mean weekly extent of the rises been 1·26 degree greater than it was. On the other hand, in the 108 *warmer* weeks it was considerably less than might have been anticipated, the deficiency being equivalent to what might have been looked for had the mean weekly extent of the rises been 1·24 degree less than it was.

Table IV exhibits the extent of operation of the rises, and of supplementary influences co-operating with or antagonising them, in the colder and warmer weeks in which the rises were associated on the one hand with increase, or on the other with decrease of sickness.

TABLE IV.—*Amount of increase of sickness when increase occurred.*

In weeks below 50° mean temperature.

Rises.	Number of weeks of increased sickness.	Extent of rises.		Value in degrees of rise of temperature of supplementary causes of increase in predominant operation.		Value in degrees of rise of temperature of united causes of increase of sickness.		Estimated increase of sickness. — Cases.		Amount of increase due to supplementary causes. — Cases.		Joint result of rises of temperature and supplementary causes of increase. — Cases.	
		Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.
Generally ..	59	271·3°	4·59°	458·54°	7·77°	729·84°	12·36°	857	14·5	1449	24·5	2306	39·08
Slight.....	16	17·1°	1·06°	111·07°	6·94°	159·82°	8·00°	54	3·3	351	22·0	405	25·3
Moderate ...	21	74·9°	3·56°	167·08°	7·95°	241·98°	11·51°	236	11·2	528	25·1	764	36·3
Considerable.	22	179·3°	8·15°	180·69°	8·21°	359·99°	16·36°	566	25·7	571	25·9	1137	51·6
In weeks above 50° mean temperature.													
Generally ...	62	226·2°	3·64°	387·97°	6·25°	614·17°	9·89°	714	11·5	1226	19·7	1940	31·2
Slight.....	24	21·9°	0·91°	218·98°	9·12°	240·88°	10·03°	69	2·8	692	28·9	761	31·7
Moderate ...	19	62·9°	3·31°	105·37°	5·54°	168·27°	8·85°	198	10·4	333	17·5	531	27·9
Considerable.	19	141·4°	7·44°	63·92°	3·36°	205·32°	10·80°	446	23·5	202	10·6	648	34·1

Amount of decrease of sickness when decrease occurred.

In weeks below 50° *mean temperature.*

Rises.	Number of weeks of decreased sickness.	Extent of rise.		Value in degrees of fall of temperature of supplementary causes of decrease in predominant operation producing manifest decrease of sickness.		Value in degrees of fall of supplementary causes of decrease *nullifying rises* and producing manifest decrease of sickness.		Estimated normal increase of sickness nullified. — Cases.		Manifest decrease of sickness produced. — Cases.		Virtual decrease of sickness. — Cases.	
		Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.
Generally ...	29	94·7°	3·26°	252·13°	8·69°	346·83°	11·95°	299	10·3	797	27·4	1096	37·7
Slight.....	12	11·3°	0·94°	139·01°	11·58°	150·31°	12·52°	36	3·0	439	36·5	475	39·5
Moderate ...	12	42·3°	3·52°	69·09°	5·75°	111·39°	9·27°	134	11·2	218	18·1	352	29·3
Considerable.	5	41·1°	8·22°	44·02°	8·80°	85·12°	17·02°	129	25·8	140	28·0	269	53·8
In weeks above 50° *mean temperature.*													
Generally ...	45	137·8°	3·06°	380·55°	8·45°	518·35°	11·51°	435	9·7	1203	26·7	1638	36·4
Slight.....	18	20·4°	1·13°	172·32°	9·57°	192·72°	10·70°	64	3·6	545	30·2	609	33·8
Moderate ...	20	61·4°	3·07°	171·82°	8·59°	233·22°	11·66°	194	9·7	543	27·1	737	36·8
Considerable.	7	56·0°	8·00°	36·08°	5·15°	92·08°	13·15°	177	25·3	115	16·4	292	41·7

FALLS OF MEAN TEMPERATURE.

In the colder weeks of the year a fall of mean temperature was much more frequently associated with decrease of sickness than in the warmer weeks; that is, in 68·93 per cent. of the colder, and in 46·66 per cent. of the warmer weeks.

The following table relates to the clear gain by way of decrease or increase of sickness.

TABLE V.—*General results of the* 103 *falls in weeks with a mean temperature below* 50°.

Falls.	No. of falls.	Total extent in degrees.	Estimated clear gain by way of decrease of sickness.	Actual clear decrease of sickness.	Number of cases of decrease above or below estimate.	Number of degrees of fall of temperature represented by the difference of estimated and actual decrease.	
			Cases.	Cases.	Cases.	Sum.	Weekly mean.
Generally ...	103	433·3°	1368	2149	+ 781	+247·15°	+ 2·40°
Slight.....	22	23·8°	75	247	+ 172	+ 54·43°	+ 2·47°
Moderate ...	49	170·8°	540	882	+ 342	+108·22°	+ 2·20°
Considerable.	32	238·7°	754	1020	+ 266	+ 84·17°	+ 2·62°

General results of the 90 *falls in weeks with a mean temperature of* 50° *and upwards.*

Generally ...	90	266·8°	843	136	— 707	—223·73°	— 2·48°
Slight.....	36	36·3°	115	...	— 420	—132·91°	— 3·69°
Moderate ...	38	119·2°	377	86	— 291	— 92·08°	— 2·42°
Considerable.	16	111·3°	352	365	+ 13	+ 4·11°	+ 0·25°

The clear gain by way of decrease of sickness in the colder weeks of fall of mean temperature was nearly sixteen times as large as in the warmer weeks. This is not sufficiently accounted for by the difference in the sum of the falls in the

two series of weeks, since the sum of the falls in the colder weeks does not amount to twice that in the warmer weeks. In the 103 colder weeks the clear gain by way of decrease of sickness was such as might have been looked for had the mean weekly extent of the falls been 2·4 degrees greater than it was, and in the 90 warmer weeks such as might have been looked for had it been 2·48 degrees less than it was.

Table VI exhibits the extent of operation of the falls of mean temperature that took place, and of supplementary co-operating and antagonistic influences in weeks in which the falls were associated on the one hand with decrease, on the other with increase of sickness.

TABLE VI.—*Amount of decrease of sickness when decrease occurred.*

In weeks below 50° mean temperature.

Falls.	Number of weeks of decreased sickness	Extent of falls.		Value in degrees of fall of temperature of supplementary causes of decrease in predominant operation.		Value in degrees of fall of temperature of united causes of decrease of sickness.		Estimated decrease of sickness. — Cases.		Amount of decrease due to supplementary causes. — Cases.		Joint results of falls of temperature and supplementary causes of decrease. — Cases.	
		Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.
Generally ...	71	297·1°	4·18°	675·63°	9·51°	972·73°	13·69°	939	13·2	2135	30·0	3074	43·2
Slight.....	14	16·1°	1·15°	168·03°	12·00°	184·13°	13·15°	51	3·6	531	37·9	582	41·5
Moderate ...	35	116·5°	3·32°	291·45°	8·32°	407·95°	11·64°	368	10·5	921	26·3	1289	36·8
Considerable.	22	164·5°	7·47°	216·13°	9·82°	380·63°	17·29°	520	23·6	683	31·0	1203	54·6
In weeks above 50° mean temperature.													
Generally ...	42	139·2°	3·31°	324·05°	7·71°	463·25°	11·02°	440	10·4	1024	24·4	1464	34·8
Slight.....	15	15·0°	1·00°	104·74°	6·98°	119·74°	7·98°	47	3·1	331	22·1	378	25·2
Moderate ...	17	53·9°	3·17°	115·18°	6·77°	169·08°	9·94°	170	10·0	364	21·4	534	31·4
Considerable.	10	70·3°	7·03°	104·43°	10·44°	174·73°	17·47°	222	22·2	330	33·0	552	55·2

Amount of increase of sickness when increase occurred.

In weeks below 50° *mean temperature.*

Falls.	Number of weeks of increased sickness.	Extent of falls.		Value in degrees of rise of temperature of supplementary causes of increase in predominant operation producing manifest increase of sickness.		Value in degrees of rise of supplementary causes of increase *nullifying falls* and producing manifest increase of sickness.		Estimated normal decrease of sickness nullified. — Cases.		Manifest decrease of sickness produced. — Cases.		Virtual increase of sickness. — Cases.	
		Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.
Generally ...	31	135·5°	4·37°	292·40°	9·43°	427·45°	13·80°	428	13·8	925	29·8	1353	43·6
Slight.....	7	7·0°	1·00°	106·01°	15·14°	113·01°	16·14°	22	3·1	335	47·8	357	50·9
Moderate ...	14	54·3°	3·83°	128·79°	9·19°	183·09°	13·02°	171	12·2	407	28·6	578	40·8
Considerable.	10	74·2°	7·42°	57·91°	5·79°	132·11°	13·21°	234	23·4	183	18·3	417	41·7
In weeks above 50° *mean temperature.*													
Generally ...	45	125·2°	2·78°	420·25°	9·33°	545·45°	12·11°	396	8·7	1328	29·5	1724	38·2
Slight.....	19	20·9°	1·10°	219·30°	11·54°	240·20°	12·64°	66	3·4	693	36·4	759	39·8
Moderate ...	20	63·3°	3·16°	141·45°	7·07°	204·75°	10·23°	200	10·0	448	22·4	648	32·4
Considerable.	6	41·0°	6·83°	59·17°	9·86°	100·17°	16·69°	129	21·5	187	31·1	316	52·6

A STUDY

OF THE

INFLUENCE OF WEATHER AND SEASON UPON PUBLIC HEALTH, &c.

III. THE INFLUENCE OF DAILY RANGE OF ATMOSPHERIC TEMPERATURE.

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The influence of variations of daily range of temperature may be studied by itself on Tables I and II accompanying this paper. The first or *primâ facie* inference that would be drawn from them is that an alteration of range, if slight (under 2 degrees) or moderate (2 and under 5 degrees) is calculated to lessen sickness, and if considerable (to the extent of 5 degrees and upwards) to increase sickness, and that the result is the same, although different in degree, whether the alteration of range be by way of increase or diminution. If, however, we take into our consideration the rise or fall of temperature accompanying these alterations of range, a somewhat different aspect is given to the results. With a view to the condensation of my remarks, however, I shall not enter fully into this part of the question, limiting myself to an inquiry into the modification which simultaneous increase or

decrease of daily range introduces into the results upon sickness of a rise and fall of mean temperature.

In Table III I have tabulated separately the weeks of rise and fall of mean temperature, distinguishing those weeks in which the rises or falls were associated with increase or with diminution of mean daily range.

I. *Upon the results of rises.*

Among the 196 weeks of rise of mean temperature tabulated in my first communication, there were 108 in which the rise was associated with an increase, and 83 in which it was associated with a diminution, of mean daily range. Generally it may be stated that when rises of mean temperature were accompanied by an increase of daily range, their normal operation was less strongly marked than when they were accompanied by a diminution of range. Thus—

For every 1000 cases of increase of sickness there occurred—

	Decrease of sickness.
In the course of 108 weeks of rise associated with *increased* range,	514 cases.
„ 83 „ *diminished* „	444 „

Now, this would be a marked result had the mean weekly extent of the rises been the same in the two series of weeks; but the result becomes more striking still when we observe that the mean extent of the rises in the former set of weeks (4·35 degrees) was greater than it was in the latter set of weeks (2·95 degrees.)

1. *As to the frequency of occurrence of increase of sickness in the two groups of weeks.*

In the weeks of rise, with increased range, increase of sickness occurred less frequently than when the range of temperature was simultaneously reduced. In the former, increase of sickness was observed in 60·18 per cent. of the weeks, and in the latter in 63·85 per cent. This result be-

Mean daily ran	E OF SICKNESS.				NEITHER INCREASE NOR DECREASE OF SICKNESS.			
	nge.	Frequency per cent. of weeks.	Amount of decreased sickness. — Cases.		No. of weeks.	Alteration of range.		Frequency per cent. of weeks.
	an.		Sum.	Mean.		Sum.	Mean.	
Altered general	8°	48·70	6557	34·8	5	16·9°	3·3°	1·29
Slightly altered	9°	50·87	2862	32·8	3	3·8°	1·2°	1·75
Moderately „	2°	51·44	2632	37·0	1	3·2°	3·2°	0·72
Considerably „	1°	38·96	1063	35·4	1	9·9°	9·9°	1·29
Unaltered	—	28·57	17	8·5	—	—	—	—

Sho ion, and fluctuations of general sickness.

Mean daily ran	SE OF SICKNESS.				NEITHER INCREASE NOR DECREASE OF SICKNESS.			
	ange) or) of	Frequency per cent. of weeks.	Amount of decreased sickness. — Cases.		No. of weeks.	Alteration of range of increase (+) or decrease (—) of sickness.		Frequency per cent. of weeks.
	ean.		Sum.	Mean.		Sum.	Mean.	
Increased gener	2·7°	48·22	2943	30·9	3	+ 5·2°	+ 1·7°	1·52
Slightly increas	0·9°	48·88	1415	32·1	2	+ 2·0°	+ 1·0°	2·22
Moderately „	3·1°	50·70	1085	30·1	1	+ 3·2°	+ 3·2°	1·40
Considerably „	6·8°	41·66	443	29·5	—	—	—	—
Decreased gener	2·9°	49·20	3614	38·8	2	— 11·7°	— 5·8°	1·05
Slightly decreas	1·0°	53·08	1447	33·6	1	— 1·8°	— 1·8°	1·23
Moderately „	3·3°	52·23	1547	44·2	—	—	—	—
Considerably „	7·5°	36·58	620	41·3	1	— 9·9°	— 9·9°	2·43

of the operation of rises and falls of mean temperature.

	REASE OF SICKNESS.					NEITHER INCREASE NOR DECREASE OF SICKNESS.					
	Alteration of range of increase (+) or decrease (—) of sickness.		Frequency per cent. of weeks.	Amount of decreased sickness. — Cases.		No. of weeks.	Extent of rises.		Alteration of range of increase (+) or decrease (—) of sickness.		Frequency per cent. of weeks.
	Sum.	Mean.		Sum.	Mean.		Sum.	Mean.	Sum.	Mean.	
R	135·3°	+ 4·1°	38·88	1141	27·1	1	3·5°	3·5°	+ 0·3°	+ 0·3°	0·92
	12·6°	+ 0·9°	37·83	402	28·7	1	3·5°	3·5°	+ 0·3°	+ 0·3°	2·70
	61·4°	+ 3·0°	42·55	621	31·0	—	—	—	—	—	—
	61·3°	+ 7·6°	33·33	118	14·7	—	—	—	—	—	—
R	74·4°	— 2·4°	36·14	842	28·0	—	—	—	—	—	—
	18·4°	— 1·0°	43·90	450	25·0	—	—	—	—	—	—
	28·3°	— 3·5°	30·76	291	36·2	—	—	—	—	—	—
	27·7°	— 6·9°	25·00	101	25·2	—	—	—	—	—	—
Ra	—	—	40·00	17	8·5	—	—	—	—	—	—

	EASE OF SICKNESS.					NEITHER INCREASE NOR DECREASE OF SICKNESS.					
	eration of range increase (+) or ecrease (—) of sickness.		Frequency per cent. of weeks.	Amount of decreased sickness. — Cases.		No. of weeks.	Extent of falls.		Alteration of range of increase (+) or decrease (—) of sickness.		Frequency per cent. of weeks.
	Sum.	Mean.		Sum.	Mean.		Sum.	Mean.	Sum.	Mean.	
Ra	115·7°	+ 2·2°	58·62	1772	34·7	2	2·7°	1·3°	+ 4·9°	+ 2·4°	2·29
	29·8°	+ 0·9°	56·60	1013	33·7	1	0·7°	0·7°	+ 1·7°	+ 1·7°	1·88
	44·4°	+ 3·1°	63·63	434	31·0	1	2·0°	2·0°	+ 3·2°	+ 3·2°	4·54
	41·5°	+ 5·9°	58·33	325	46·4	—	—	—	—	—	—
Ran	196·7°	— 3·1°	59·61	2766	44·6	2	0·4°	0·2°	— 11·7°	— 5·8°	1·92
	25·8°	— 1·0°	62·50	997	39·8	1	0·3°	0·3°	— 1·8°	— 1·8°	2·50
	86·1°	— 3·3°	65·00	1250	48·0	—	—	—	—	—	—
	84·8°	— 7·7°	45·83	519	47·1	1	0·1°	0·1°	— 9·9°	— 9·9°	4·16
Ran	—	—	—	—	—	—	—	—	—	—	—

comes the more striking when it is observed that both the mean extent of the rises of temperature and of the alteration of range was greater in the former than in the latter group of weeks.

In the weeks of slight alteration of range, however, the frequency of increase of sickness was greater when it was by way of increase than when it was by way of diminution; in the former it occurred in 59·45 per cent. of the weeks, and in the latter in only 56·09 per cent. But then it is to be observed that extent of the rises was greater in the former than in the latter group of weeks.

In the weeks of moderate and considerable increase of range the frequency of occurrence of increase of sickness was much less than in those weeks when the rises were associated with a lessening of the range; thus—

In the weeks of moderately increased range increase of sickness occurred in 57·44 per cent. of the weeks, and in those of moderately diminished range in 69·23 per cent. Yet the mean extent of the rises in the former group of weeks was 4·29 degrees, and in the latter only 2·92 degrees.

In the weeks of considerably increased range increase of sickness occurred in 66·66 per cent. of the weeks, and in those of considerably diminished range in 75·0 per cent. Yet the mean extent of the rises in the former group of weeks was 5·59 degrees, and in the latter only 4·15 degrees.

2. *As to the clear gain by way of increase of sickness in the two groups of weeks.*

Deducting the cases of decrease from those of increase of sickness in the two groups of weeks, and taking a mean of the weekly clear gain by way of increase of sickness, we find that, on the whole, in those weeks of rise of mean temperature which were accompanied by an increased daily range the normal operation of the rises was less in amount than in those weeks in which the range was simultaneously lessened; the former gave a clear mean weekly gain of 9·9 cases, while the latter gave a clear gain of 12·7 cases per week.

It was only in the cases of *slight* alteration of range that an opposite result was obtained.

Taking now further into our consideration the mean extent of the rises of temperature in the two groups of weeks, it is worthy of observation that throughout each series the corresponding weeks of increased range presented a greater mean rise than those of diminished range. Normally, this might have been expected to have resulted in a greater clear gain by way of increase of sickness in the former than in the latter group throughout. That the actual result was the reverse of this serves greatly to strengthen the inference drawn on the first view of the Table.

The following Table exhibits more fully the apparent operation of increase or diminution of range in association with the question of the extent of the rise of temperature.

TABLE IV.—*General results where rises of mean temperature were associated with increase or decrease of mean daily range.*

	Number of weeks.	Total extent of rises in degrees.	Estimated clear gain by way of increase of sickness.	Actual clear gain by way of increase of sickness.	Number of cases of increase above or below estimate.	Number of degrees of rise of temperature represented by the difference of estimated and actual increase.	
			Cases.	Cases.	Cases.	Sum.	Weekly mean.
Range increased generally.	108	469·8°	1484	1077	— 407	—128·79°	— 1·19°
slightly increased ...	37	133·8°	423	393	— 30	— 9·49°	— 0·25°
moderately „ ...	47	201·8°	638	256	— 382	—120·88°	— 2·57°
considerably „ ...	24	134·2°	424	428	+ 4	+ 1·26°	+ 0·05°
Range decreased generally.	83	246·9°	780	1051	+ 271	+ 85·75°	+ 1·03°
slightly decreased ...	41	104·1°	329	56	— 273	— 86·39°	— 2·10°
moderately „ ...	26	76·1°	230	650	+ 420	+132·91°	+ 5·11°
considerably „ ...	16	66·7°	211	345	+ 134	+ 42·40°	+ 2·65°

3. *As to the amount of increase of sickness, when increase occurred, in the two groups of weeks.*

In the 65 weeks of rise with increased range, in which increase of sickness actually occurred, the mean amount of the increase was 34·1 cases; while in the 53 weeks accompanied by diminished range the mean amount of the increase of sickness was 35·7 cases. This difference, slight at the first glance, becomes more important when it is observed that in the 65 weeks the mean extent of the rises of temperature was 4·61 degrees, while in the 53 weeks it was less, namely, 3·48 degrees.

4. *As to the amount of decrease of sickness, when decrease occurred, in the two groups of weeks.*

In the 42 weeks of rise with increased range, in which decrease of sickness took place, the mean amount of the decrease was 27·1 cases; while in the 30 weeks of rise with diminished range it was 28 cases. This is a trifling difference, but it acquires more importance when it is observed that the mean extent of the rises (3·96 degrees) in the former set of weeks was nearly twice as great as in the latter (2·07 degrees). Viewing this difference in the extent of the rises by itself, we should have anticipated a much greater difference in the amount of the decreases of sickness when they occurred. That the difference thus anticipated did not exist must be attributed to the operation of the increased range assisting the other causes of decrease in the 42 weeks of rise, and to the operation of the lessened range antagonising the causes of decrease in the 30 weeks of rise.

II. *Upon the results of falls.*

Among the 193 weeks of fall of mean temperature formerly tabulated there were 87 in which the fall was as-

sociated with an increase, and 104 in which it was associated with a diminution, of mean daily range.

As with rises, so too with falls, it may be stated generally that the normal operation of the falls upon fluctuations of sickness was, on the whole, less strongly marked when they were accompanied by an increase than when they were accompanied by a diminution of daily range. Thus—

For every 1000 cases of decrease of sickness there occurred—

		Increase of sickness.
In the course of 87 weeks of fall associated with	*increased*	range, 555 cases.
„ 104 „	*diminished*	„ 430 „

The mean extent of the falls was not very different in the two groups of weeks, being 3·55 degrees in the one and 3·70 degrees in the other.

1. *As to the frequency of occurrence of decrease of sickness in the two groups of weeks.*

In the weeks of fall with an increased range decrease of sickness occurred rather less frequently than when the range of temperature was diminished. In the former, decrease of sickness was observed to occur in 58·62 per cent. of the weeks, while in the latter it occurred in 59·61 per cent. The difference is trifling. Taking into account the different extent of the alteration of range in the several weeks, it is to be observed that in the 12 weeks of considerably increased range the frequency of occurrence of decrease of sickness was greater, on the whole, than in the 24 weeks of considerably diminished range. On making a further inquiry into this apparent anomaly, however, it is found that it depends upon a difference in the results of considerably altered range when associated with falls in the colder and warmer seasons of the year. In the 7 weeks with a mean temperature below 50°, in which the range was considerably increased, decrease of sickness occurred in 71·42 per cent. of the weeks, while in the 9 corresponding weeks, with considerably diminished

range, decrease of sickness occurred in only 44·44 per cent., notwithstanding that the mean extent of the falls in the latter set of weeks was nearly twice as great as in the former. On the other hand, the result in the warmer season, in weeks with a mean temperature of 50° and upwards, was more normal; in the 5 weeks of considerably increased range decrease of sickness occurred in 40 per cent., and in the 15 weeks of considerably diminished range in 46·66 per cent.

2. *As to the clear gain by way of decrease of sickness in the two groups of weeks.*

Deducting the cases of increase from those of decrease of sickness in the two groups of weeks, and taking a mean of the weekly clear gain by way of decrease of sickness, we find that, on the whole, in those weeks in which a fall of temperature was accompanied by an increased daily range, the normal operation of the falls was much less in amount than in those weeks in which the range was simultaneously lessened; the former gave a mean clear weekly gain, by way of decrease of sickness, of 9·0 cases, the latter of 15·1 cases. But here again we observe that, while in the weeks of slight and moderate alteration of range the general rule was followed, it was otherwise in the weeks of considerable alteration of range. In these weeks the clear gain was greater where the range was increased than in those where it was diminished. Here also again we find the anomaly is to be referred to a difference of results in the cooler and warmer seasons of the year. In the colder seasons, in those weeks in which the mean temperature was below 50°, the mean clear gain by way of decrease of sickness was 29·8 cases in the weeks of considerably increased range, and only 8·7 cases in those of considerably diminished range, notwithstanding that in the latter weeks the mean extent of the falls was very much greater than in the former weeks. On the other hand, in the warmer season of the year, in the weeks with a mean temperature of 50° and upwards, there was

actually a clear gain of increase of sickness in the weeks where the range was considerably increased to the mean extent of 8 cases; while there was a clear gain, by way of decrease, to the mean extent of 11·4 cases in those weeks in which the daily range was lessened.

The following Table exhibits more fully the apparent operation of increase or diminution of range in association with the question of the extent of the falls of temperature.

TABLE V.—*General results where falls of mean temperature were associated with increase or decrease of mean daily range.*

	Number of weeks.	Total extent of falls in degrees.	Estimated clear gain by way of decrease of sickness.	Actual clear gain by way of decrease of sickness.	Number of cases of decrease above or below estimate.	Number of degrees of fall of temperature represented by the difference of estimated and actual decrease.	
			Cases.	Cases.	Cases.	Sum.	Weekly mean.
Range increased generally.	87	309·2°	977	788	— 189	— 59·81°	— 0·68°
slightly increased...	53	199·0°	629	380	— 249	— 78·79°	— 1·48°
moderately „ ...	22	83·0°	262	239	— 23	— 7·27°	— 0·33°
considerably „ ...	12	27·2°	86	169	+ 83	+ 26·26°	+ 2·18°
Range decreased generally.	104	385·3°	1218	1575	+ 357	+112·97°	+ 1·08°
slightly decreased...	40	108·9°	344	448	+ 104	+ 32·91°	+ 0·82°
moderately „ ...	40	171·3°	541	877	+ 336	+106·32°	+ 2·65°
considerably „ ...	24	105·1°	332	250	— 82	— 25·94°	— 1·08°

3. *As to the amount of decrease of sickness, when decrease occurred, in the two groups of weeks.*

In the 51 weeks of fall with increased range, in which decrease of sickness actually occurred, the mean amount of the decrease was 34·7 cases; while in the 62 weeks accompanied by diminished range the mean amount of the

decrease was 44·6 cases. There was very little difference in the mean extent of the falls in these two groups of weeks. Taking the extent of the falls into consideration, however, in those weeks in which the alteration of range was 5 degrees and upwards, the mean decrease in the weeks of increased range was somewhat larger than in those of diminished range. On further inquiry we find that this depends on a difference in the kind of result in the colder from that observed in the warmer season of the year. In the colder season (mean temperature below 50°) the mean decrease of sickness in the weeks of considerably increased range was 53·2 cases, while it was 51·5 cases in the weeks of considerably lessened range, notwithstanding that the mean extent of the falls was greater. In the warmer season, however, the mean decrease in the weeks of considerably increased range was 29·5 cases, and in those of considerably lessened range 44·7 cases.

4. *As to the amount of increase of sickness, when increase occurred, in the two groups of weeks.*

In the 34 weeks of fall with increased range, in which increase of sickness occurred, the mean amount of the increase was 28·9 cases; while in the 40 weeks of fall with diminished range it was 29·7 cases. The virtual difference, when the extent of the falls in the two groups of weeks is taken into consideration, is somewhat greater than this. Now if, as appears to be true, a diminished range is opposed to an increase of sickness in weeks of fall of temperature, we must recognise here a concentration of the other supplementary causes of increase of sickness in the 40 weeks of diminished range. This concentration chiefly took place in the weeks in which the range was considerably altered. This does not appear at first sight; but it comes out distinctly on calculating the force of the influence exerted by the falls, varying in extent, in each of the subordinate groups of weeks. Thus, in the 22 weeks of slightly increased range the antagonistic force opposed to the falls equalled that normally exerted by 20 degrees of rise, while in the 14 weeks of slightly

diminished range by 19·1 degrees of rise; in the 7 weeks of moderately increased range it equalled that exerted by 21·9 degrees of rise, and in the 14 weeks of moderately diminished range by 20·7 degrees of rise. On the other hand, in the 5 weeks of considerably increased range the antagonistic force opposed to the falls equalled that normally exerted by only 13·0 degrees of rise, while in the 12 weeks of considerably diminished range it equalled that exerted by 22·4 degrees of rise. Again, it comes out, on pursuing the inquiry, that while the normal influence of the alteration of range was exerted in the warmer season of the year, it was otherwise in the colder season, since with a mean extent of falls of 1·45 degree the mean increase of sickness in 2 weeks was 28·5 cases, in the warmer season with a mean fall of 6·80 degrees it was nearly the same, namely, 25·4 cases.

I may state here that I have found, by pursuing the inquiry into the influence of daily range upon sickness by a totally different method, that its operation in the colder season of the year is very much less than in the warmer season, being quite subordinate to that of the temperature, while in the warmer season the reverse is the case.

The diagrams published with my first communication illustrate both this and the preceding paper.

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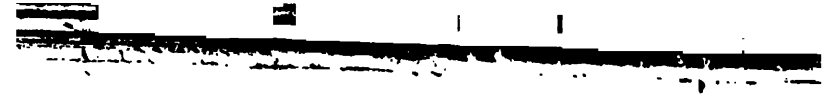
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